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TEXAS State Journal of Medicine

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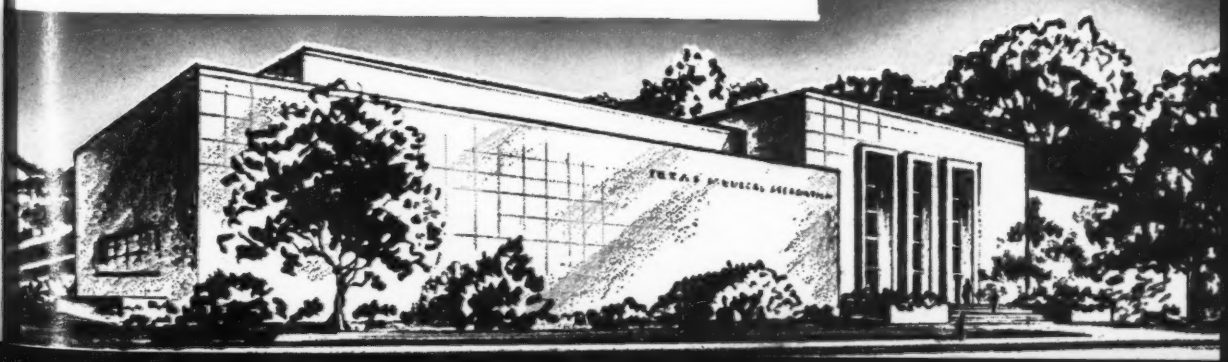

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President's Page

VETERANS' MEDICAL CARE

The greatest internal threat for the socialization of medicine in the United States today lies in the demands of pressure groups on Congress to enact laws providing nonservice connected medical care for war veterans in Veterans Administration hospitals. There is no question concerning provision of the highest type of medical care for service connected disabilities under government supervision at taxpayers' expense, but the motives of organizational leaders who attempt to make political capital of the misfortunes of heroic veterans by speaking a desire to "do right by our boys" are difficult to discern. The majority of veterans are self-sufficient, intelligent, taxpaying citizens and did not serve their country to become government wards.

The idea of expanding veterans' health care into a blanket free-for-all program was at top political pitch during closing days of the last Congress, but no essential changes affecting the care of nonservice connected disabilities in VA hospitals were made. Chairman Bernard W. (Pat) Kearney (R., New York) of the House's Veterans Affairs subcommittee asked that veterans' organizations make recommendations on the issue "after their conventions this fall." Among questions the committee raised were: What is the definition of service connected disability? How far should Congress go beyond the present GI Bill of Rights in saying what government owes in nonservice connected cases? Would enforcement of existing inability-to-pay provisions for nonservice connected cases be preferable to new legislation?

Officials from General Accounting Offices in investigating forty-six VA hospitals found instances of veterans who had incomes ranging from $4,000 to $50,000 a year, some having real property and other assets between $20,000 and $500,000, receiving free hospitalization and medical care.

The Veterans of Foreign Wars convention August 2 adopted a resolution recommending the strengthening of existing legislation. The VFW advocates investigation of the applicant's financial status by the VA contact officer assisted by officials of the VFW. The Disabled American Veterans and AMVETS hold essentially these same views. The American Legion, however, on September 2 expressed a different

viewpoint: "... the man or woman who was called into the Armed Forces ... has been placed in a very special class by that Government. The United States, through our Congress, have always considered all veterans a special class of citizens and have always shown respect and affection for these veterans by certain benefits and just compensations ... veterans ... are indeed a distinct class and the direct and specific responsibility of our Federal Government when in need, or are sick, broke and have no place to go."

Dr. Walter B. Martin, President-Elect of the American Medical Association on July 13 before Congress outlined the AMA's opposition to further treatment in VA hospitals of veterans with nonservice connected disabilities, other than tuberculosis or neuropsychiatric disorders. He made it clear that Congress should enact legislation limiting medical and hospital care for veterans in VA and other federal hospitals to (1) veterans with peacetime or wartime service whose disabilities or diseases are service connected and (2), within limits of existing facilities, to veterans with wartime service suffering from tuberculosis or neuropsychiatric disorders of nonservice origin who are unable to pay for hospitalization. He further proposed that (1) care for remaining veterans be discontinued and their responsibility revert to the individual or the community and (2) Congress reanalyze the entire question of whether the chronically ill veteran with nonservice connected disability is a federal or local responsibility.

The AMA House of Delegates has recommended that every effort be made by national, state, and local medical societies to inform the profession and the public concerning the nature of the problem, position of the AMA, and reasons on which that position is predicated. To implement these recommendations the Committee on Federal Medical service of the Council on Medical Service sponsored a meeting September 1 in Chicago. Plans for seven regional meetings were made, one to be held November 6 at Dallas with representatives from the Texas, Louisiana, Oklahoma, Arkansas, and Mississippi medical associations participating. Before these regional meetings are held, appointment of a committee on veterans' medical care by each state association and each county society is recommended.

George Tunnell M.D.

PR diagnotes

REPORT OF PUBLIC RELATIONS COMMITTEE: The public relations program for 1954 is now in the mill under the guidance of the Committee on Public Relations. Projected action seems to fall along the following lines: to emphasize again both internal and external relationships of the profession. . . . with some changes of stress--a few additions.

On the internal side: Continue to stress increasing of society interest and attendance - state and county levels, strengthening lines of communication to PR chairmen and individual doctors - increasing Auxiliary contacts, cooperation. Two additional stresses, one new project added. Particular attention to be given to securing best possible annual session speakers, building interest in PR Diagnotes. By including this page in the Journal, it is hoped to encourage wider reader-audience. New project is "Medical Students Day" - more below.

On the external side: Move into the broadening of four-point program of last year. "Your Medical Dollar's Worth" to be the central theme including sub-headings "Cost of Medical Care," "Availability of Around-the-Clock Medical Service," "Doctor Distribution," "Front Office Technique," and "Miscellaneous." Mediation committee to be stressed and a promotion kit, "Your Medical Dollar's Worth," to be distributed to all societies. Continued stress on emergency telephone service under "Around-the-Clock" - a new, tremendously popular project added - "Health Forums." Secretary training kit has created keen interest - should extend this to course outlines due soon.

After success of Dallas "Medical Students Day" last month, plans for additional programs forthcoming rapidly. Next to be held - Baylor University College of Medicine, Houston. Plans now say sometime early December. Incidentally, students of Southwestern Medical School high in praise of Dallas meeting. Opinions came in on evaluation card sent out by committee - some good suggestions, too.

Special newspaper issues worth mentioning again - just once more. Success amazing - covered the entire state. Circulation managers of all papers have been asked to send copies to all county societies.

Woman's Auxiliary handbook highly successful - was presented in rough last month at Executive Board meeting at Association headquarters. Should be published in final form by this reading - contains valuable information for county and state officers, committee chairmen - a must for officers-elect before taking office.

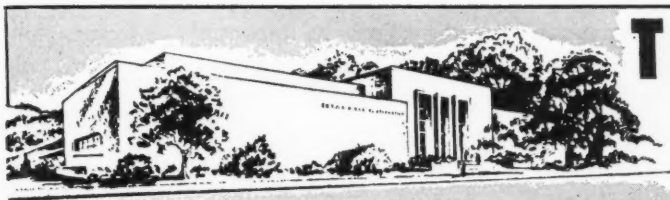
ON THE NATIONAL SCENE: From AMA Washington News Letter, No. 36 - "Amid the sizzling heat here, the National Advisory Committee to Selective Service announcement on drafting of doctors came as a refreshing breeze. The committee says it doesn't expect any more calls for physicians for about the next twelve months. Of course, this does not mean there will be no call-ups if the world situation deteriorates or the political talks over the future of Korea break down. It's simply the committee's studied view of the doctor draft status for the next year."

Forthcoming clinical session of the American Medical Association should be kept in mind--St. Louis, December 1-4. Of special interest: the sixth annual National Medical Public Relations Conference will be held November 30, immediately preceding the AMA session, at St. Louis. Medical society officers, executive secretaries, and PR personnel are particularly invited.

FROM OVER THE STATE: Texas Academy of General Practice meeting in Dallas, September 21-23, very successful - press coverage encouraging - attendance fine - program highly informative.

Dallas County Society's preceptorship lectures being held at Baylor Hospital, first Tuesday of each month - reports say progressing in fine manner. Subjects include Public Relations, Medical Ethics and Medical Economics, County Medical Society, General Medical Organization, Medical Practice Act, Malpractice, Coroner-Narcotic-Compensation Laws. Some other societies are finding similar lecture series valuable for newer members.

Congratulations are due San Antonio, Fort Worth, and Austin doctors. During the past month the Bexar County Medical Society celebrated its one hundredth anniversary, and the Fort Worth Academy of Medicine opened its new building, simultaneous with the fiftieth anniversary of Tarrant County Medical Society. This month, the Travis County Medical Society will observe its centennial.



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Volume 49 **Austin, Texas, October, 1953** **Number 10**

HOSPITALS-INSURANCE-PHYSICIANS COMMITTEE

A hard working committee which cuts across three fields of interest and collaborates in solving the problems pertinent to all three has been active without fanfare since 1952. On August 16, 1952, in Dallas, the Hospitals-Insurance-Physicians Joint Advisory Committee of Texas was organized, and bimonthly meetings have been held since that time.

The Texas Medical Association, Private Clinics and Hospitals Association of Texas, Texas Hospital Association, Texas Association of Accident and Health Underwriters, Texas Accident and Health Claims Underwriters Association, and Health and Accident Underwriters Conference were represented in conferences held in 1950 and 1951 to develop a standardized hospitalization and physician's insurance report form. The success of this form, the first of which was drafted in June, 1951, and which in revised form has been adopted rather generally throughout the state, together with an awareness of many other problems of mutual interest and importance led to the establishment of the permanent committee.

Objectives of the H-I-P Committee have been listed thus:

1. To discuss and counsel in open meeting all the problems which affect the administrative functions of each group (a) wherein they overlap, (b) wherein they trespass on the rights or prerogatives of another, and (c) wherein the administrative functions of one group may cause unwarranted economic distress to another or impair its prestige.

2. Through joint counsel (a) to prevent the initiation or development of practices which in the future might impair the proper functioning of any of the group and (b) to promote harmony within all groups.

3. To act as a public relations committee (a) between each group and the general public and (b) as a unit against governmental interference or encroachment upon the groups' business or professional prerogatives.

4. To act as a grievance body in cases wherein (a) major violation by partisans of one group impair harmony of all, (b) action of partisans of one group do anything to bring public disapproval, and (c) actions of partisans of one group commit or may commit fraud upon the public.

In addition to development of the standard hospitalization and physician's insurance report

form, the cooperative action of the committee has resulted in joint endorsement of a standard group hospitalization insurance report form, an exchange of speakers, and efforts to assist the department of agricultural education at Texas Agricultural and Mechanical College in preparing a teaching plan for rural schools.

Drs. T. C. Terrell, Fort Worth, and Everett C. Fox, Dallas, currently represent the Texas Medical Association on the H-I-P Committee. Another physician, Dr. Harvey Renger, Hallettsville, is on the committee as a representative of the Private Clinics and Hospitals Association of Texas.

Undoubtedly one of the soundest methods of bringing to light sources of friction between business and professional groups and of solving problems which relate not only to those groups but to the public as well is the establishment of such committees as the Hospitals-Insurance-Physicians Joint Advisory Committee. It already has demonstrated its usefulness, and there is every reason to assume that it will continue to iron out differences between the interests represented in its membership and to promote advancements in the area of better hospitalization and medical care insurance.

WELCOME ABOARD!

The decision of the Board of Trustees, made during the September Executive Council meeting in Austin, that senior students at the three Texas medical schools will receive complimentary copies of the TEXAS STATE JOURNAL OF MEDICINE adds a significant readership group.

Although numerically the approximately 300 new readers may not seem impressive, it might be pointed out that the future physicians who will be affected by this new policy can contribute something to the JOURNAL, as well as receive its benefits. At a time when interest in research and learning is high, when youth lends enthusiasm and new ideas to worth-while projects, students, by expressing their dislikes and "druthers," may be responsible, at least to some extent, for injecting into the Association's publication greater "vim, vigor, and vitality."

So to the new student readers of our JOURNAL, we say, "Glad to have you aboard!"

EFFECTIVE UTILIZATION OF HEALTH MANPOWER

In the September issue of the JOURNAL, editorial mention was made of the serious medical personnel shortages in existence today.

One of the latest groups to release a statement pertinent to the problem is the Health Resources Advisory Committee of the Office of Defense Mobilization. This committee expressed the viewpoint that, since shortages do exist and probably will continue to exist for some time, better utilization of available health manpower has taken on an added importance.

The committee recently forwarded a preliminary statement to each administrator and chief of the medical staff of all hospitals registered by the American Medical Association. Although the report is aimed primarily at alleviating the critical hospital nursing needs of this country, the approaches cited to bring about more effective and economical use of personnel may well be worth consideration for application to all fields of medical care.

All hospital heads are advised to analyze existing hospital procedure and routine. In doing so, it is believed that routines which have become unnecessary because of new medical technology will be apparent. Other suggestions mentioned include the use of generally accepted management techniques, recognition of the importance of job satisfaction, development of leadership skills, reallocation of work among the various manpower groups, and carrying on of continuous job analysis surveys.

What part does each physician play in better hospital personnel utilization? The committee emphasizes the fact that since physicians are responsible for the work load of the hospital,

they can do much to help achieve and promote better utilization. It has been suggested that physicians should review orders for patients daily, discontinuing and avoiding unnecessary ones and making definite distinction between emergency and routine orders. Also, that they should facilitate prompt discharges and admissions according to hospital policy, recognize the economy of scheduling hospital rounds, treatment, and surgical procedures to fit into essential hospital routine, complete medical records promptly, teach patients the advantage of self care as a part of early ambulation and rehabilitation, and cooperate with the standardizing of supplies and equipment whenever possible.

The Health Resources Advisory Committee plans to study the problem further and to collect, analyze, and disseminate available knowledge. A handbook made up of a series of pamphlets, each dealing with one specific phase of utilization of hospital personnel, is being prepared. The committee intends to make similar studies in such fields as home care and public and industrial health.

The problem is not a new one. It is, however, one which deserves and needs further study by all medical groups.

OPEN DOOR TO GOOD WILL

The new building of the Texas Medical Association has been in use slightly more than a year. During that time, at the recommendation of the Board of Trustees, the facilities of the building have been available for meetings of units of the Association and its Woman's Auxiliary, of organizations made up of members of the medical profession, and of groups allied with medicine or having scientific, educational, or cultural objectives.

From early September, 1952, immediately following the move of the central offices and Library of the Association into the new building on Lamar Boulevard in Austin, until September 15, 1953, 38 meetings exclusive of those of the Texas Medical Association or Auxiliary were held in the building. These included 28 meetings of groups nonmedical in nature or having no integral relationship with the county-district-state framework of medical organization. In addition to these 38 meetings, which drew roughly 2,300 persons, the Executive Council of the Texas Medical Association met three times and the Executive Board of the Woman's Auxiliary to the Texas Medical Association met once, adding another 500 or so persons to those who have enjoyed the meeting rooms during the first year of the building. These figures do not include the multitude of committee, council, and board meetings held throughout the twelve months.

In planning the headquarters and Library building, it was the desire of the Trustees and Building Committee to provide not only adequate working space for the staff and storage space for records and reference materials but also a "home base" for members of the Association and a means of developing good will among those who should be their friends. Although mere figures cannot demonstrate conclusively the success or failure of such a goal, the continuous use of the building facilities is indicative of some measure of achievement. The interest and gratitude of those who have met in the building, even though it cannot be totaled on an adding machine, has helped to verify that impression.

Student AMA Convention on Film

A documentary-type sound film taken at the third annual convention of the Student American Medical Association last June in Chicago is now available to county medical societies as well as medical schools. The film is to be shown to local student chapters throughout the country during the school year to give a comprehensive picture of the convention, with both business and entertainment features pictured. Application for the film may be made to the AMA's Committee on Medical Motion Pictures, 535 North Dearborn Street, Chicago 10.

Bibliographies in selected fields of medical study will be prepared by the staff of the Texas Medical Association Library for members of the Association. In addition to the references lists, most articles and books cited will be loaned without charge. Requests should be addressed to the Library at 1801 Lamar Boulevard, Austin.

ORIGINAL ARTICLES

Glucose in the Development and Treatment of Diabetic Acidosis

DONALD W. SELDIN, M. D., Dallas, Texas

So profound is the impact of ketosis on the internal environment of patients in diabetic acidosis that for many years the major studies of electrolyte metabolism were devoted to an elucidation of the effects of ketonemia and ketonuria on the volume and composition of the extracellular fluids and the excretion of water and electrolytes. It was early appreciated that ketosis represented not a primary expression of insulin lack, but rather a secondary consequence of the failure of the organism to dispose properly of carbohydrate. When ketone production finally exceeded ketone utilization, an acidosis ensued because of the decomposition of bicarbonate by invading ketone acids. Finally, as the capacity of the renal tubules to reabsorb ketones was exceeded, an intense ketonuria supervened, sweeping sodium as well as water into the urine. This sequence of events, with ketosis as the causal focus, was thought to explain not merely the profound acidosis so characteristic of this state but the polyuria and salt loss as well.

In 1933 Atchley, Loeb, and their associates[1] demonstrated that sudden withdrawal of insulin from patients with severe diabetes resulted in an abrupt acceleration of urinary losses of salt and water. It is especially noteworthy that this diuresis occurred in 1 of their patients who developed severe hyperglycemia and glycosuria, but no significant ketosis. Apparently, factors other than ketonuria were operative. In 1937 Kerpel-Fronius[5] proposed, on the basis of his own studies as well as those of others, that the profound dehydration and salt depletion characteristic of diabetic acidosis might be more intimately linked with hyperglycemia and glycosuria than with ketosis. His studies were discounted,[2] however, and for the succeeding decade the role of hyperglycemia in diabetic acidosis was evaluated largely in terms of the problem of whether or not high levels of blood sugar actually accelerate the peripheral utilization of carbohydrate.[6, 7] No attention was paid to the profound impact of glucose on the metabolism of water and electrolytes.

The following studies of the effects of hyperglycemia on the internal exchanges and urinary excretion of water and electrolytes were conducted to appraise better the role of glucose in the development and treatment of diabetic acidosis.

From the Department of Internal Medicine, Southwestern Medical School of the University of Texas.

Read before the Section on Internal Medicine, Texas Medical Association, Annual Session, Houston, April 29, 1953.

GLUCOSE IN NORMAL SUBJECTS

The injection of massive amounts of glucose into normal human subjects permits an evaluation of the impact of hyperglycemia and glycosuria when ketosis is absent.[8] The sequence of events is depicted in figure 1. After a control period during which urine was collected, blood was drawn and 700 cc. of 25 per cent glucose was injected intravenously in forty-five minutes (period A, fig. 1). The blood sugar level rose from an initial value of 108 mg. per 100 cc. to a final value of almost 800 mg. per 100 cc. immediately after the cessation of the infusion. With the development of hyperglycemia, the concentration of serum sodium fell sharply, from an initial value of 132 milliequivalents per liter to 115 milliequivalents per liter at the end of the infusion. At the same time extracellular volume (estimated from changes in the chloride space) expanded by 1 L.

This fall in the concentration of serum sodium and expansion of extracellular volume was too large to be ascribed to dilution by injected water. Increased urinary losses of sodium and water could not account for the changes, since the quantities of sodium excreted were too small and its concentration was distinctly lower in the urine than in the serum. Moreover, accelerated urinary excretion would tend to diminish extracellular volume, whereas it was actually expanded. Therefore, the reduction in the concentration of serum sodium and the concomitant expansion of extracellular volume must be due to a withdrawal of water from cells in response to the increase in the osmotic pressure of the extracellular fluid caused by high concentrations of glucose. This transfer of water from cells to extracellular fluid could not have occurred if glucose traversed cellular membranes freely, because it then would have elevated the effective osmotic pressure on both sides of the membrane equally and could not have affected the distribution of water. Hence, free glucose must be confined largely

to the extracellular fluid, probably penetrating cell membranes not by diffusion but by an active process involving its metabolic transformation. Such an interpretation is in accord with the demonstration that radioactive glucose has a volume of distribution approximating extracellular volume.

If the reduction in the concentration of serum sodium and the expansion of extracellular volume is mainly the result of dilution of extracellular fluid with water withdrawn from cells by high concentration of glucose, it might be anticipated that when carbohydrate is burned and hyperglycemia recedes, water would return to cells, resulting in a rise in serum sodium content toward normal values. This is precisely what happens (period B, fig. 1). As hyperglycemia

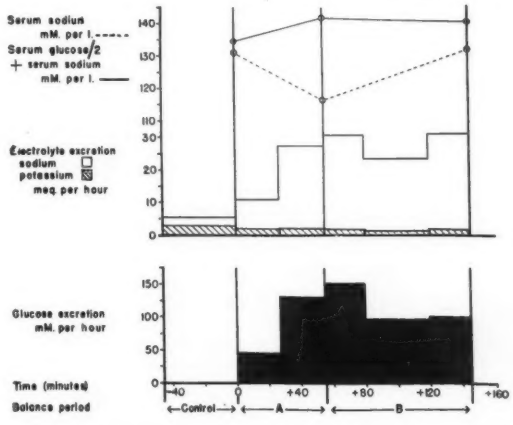

FIG. 1. Chart showing the effect on urine and serum electrolytes of the intravenous injection of 700 cc. of 25 per cent glucose in a normal human subject. Note that as massive glycosuria supervenes, sodium excretion mounts in a parallel fashion, but potassium excretion is not appreciably affected. The concentration of serum sodium falls as extreme hyperglycemia develops (period A) but rises toward normal when hyperglycemia recedes (period B) despite continued urinary losses of sodium. (After Seldin, D. W., and Tarail, R.: Effect of Hypertonic Solutions on Metabolism and Excretion of Electrolytes, Am. J. Physiol. *159*:160-174 [Oct.] 1949.)

recedes with the rapid burning of carbohydrate, the effective osmotic pressure of the extracellular fluid falls, and water returns to body cells. As a consequence, the concentration of serum sodium rises toward normal.

Studies such as these permit several conclusions: glucose does not penetrate body cells freely by diffusion; hyperglycemia therefore causes a rise in the effective osmotic pressure of the blood, drawing water out of body cells into the extracellular fluid; this results in profound cellular dehydration; at the same time the concentration of sodium and chloride in the blood falls drastically, not because sodium is lost into the urine, but because extracellular fluid is being diluted with cell water.

Synchronous with these changes in the internal environment, the excretion of salt and water into the urine rises sharply. The increase in sodium excretion is apparent from figure 1 (periods A and B), where a marked sodium diuresis has developed with hyperglycemia and glycosuria. At the same time, urine flow is markedly accelerated. Indeed, although glucose loads accelerate the renal losses of both salt and water, the losses of water are far in excess of the losses of salt, and one might anticipate that the concentration of sodium in the blood would have risen. As has been pointed out, however, this effect is masked by the capacity of glucose to draw water out of cells and thus dilute the serum sodium to values below normal.

Finally, it should be noted that, despite profound increases in the excretion of water and salt, the excretion of potassium actually decreases. Yet, the concentration of potassium in the serum falls. On the basis of balance studies, it can be shown that this fall in serum potassium is due not to urinary losses, but to a movement of potassium from extracellular fluid into cells in association with the accelerated utilization of carbohydrate.

ELECTROLYTE METABOLISM IN DIABETIC ACIDOSIS

It should be reemphasized that these studies of the osmotic effects of glucose have been conducted in normal subjects to elucidate the effects of hyperglycemia and glycosuria apart from ketosis. If we turn now to the patient with diabetic acidosis, it may be possible better to understand and hence to treat the profound distortions of electrolyte metabolism so characteristic of this state.[9] In table 1 some of the electrolyte aberrations are listed.

TABLE 1.—*Electrolyte Disturbances in Diabetic Acidosis.*

Disturbances in salt and water metabolism:
Diminished volume of the extracellular fluids.
Diminished concentrations of serum sodium and chloride.
Hypertonicity of the extracellular fluid with extreme cellular dehydration.
Disturbance in potassium metabolism:
Profound depletion of total body stores of potassium.
Elevation in the concentration of serum potassium initially; low serum potassium level developing during treatment.
Phosphorus metabolism:
Profound depletion of total body stores of phosphorus.
Elevation in the concentration of serum phosphorus initially; low serum phosphorus level developing during treatment.
Ketonemic acidosis.

From a clinical standpoint drastic salt and water depletion is the most important of the electrolyte disturbances, since this leads to peripheral vascular collapse and perhaps death. Although the magnitude of the reduction in extracellular fluid will vary with the severity of the disease and the size of the patient, deficits of about 3 L. noted in balance studies[9] emphasize the enormous depletion of extracellular volume in the average patient. In the production of this salt

depletion, vomiting and ketonuria play an important, though subsidiary, role. From the studies on normal subjects, it appears that the major part of the salt deficit arises as a consequence of hyperglycemia and glycosuria, which sweeps salt as well as water into the urine in massive amounts. Figure 2, which charts the relationship between urine flow and glucose excretion in 11 patients with diabetic acidosis, indicates that urine flow is a linear function of the rate of glucose excretion. In figure 3, where repeated injections of hypertonic glucose were administered to a patient with diabetic acidosis, a marked acceleration of sodium and chloride excretion could be elicited. This capacity of glucose to sweep salt and water into the urine is particularly dangerous, inasmuch as it is not inhibited by the desperate salt depletion and dehydration characteristic of diabetic acidosis.

The low concentration of sodium and chloride in the serum long have been puzzling. Although it was long recognized that the total amount of these substances in the extracellular fluid was greatly diminished, it was not clear why their concentrations should be depressed. It should be noted that the fluid lost during the development of diabetic acidosis is hypotonic with respect to sodium chloride. The urine formed contains sodium at a concentration considerably below that in serum because glycosuria accelerates to a greater extent the excretion of water than of salt. Insensible perspiration and sweat are both hypotonic; hyperventilation results in the loss of water alone. These losses of water in excess of salt by themselves would result in an elevated concentration of serum sodium and chloride, but this is rare in diabetic acidosis. Probably cellular water, drawn into the extracellular compartment by the increased effective osmotic pressure resulting from hyperglycemia, dilutes the concentration of sodium and chloride to normal or more often low values.

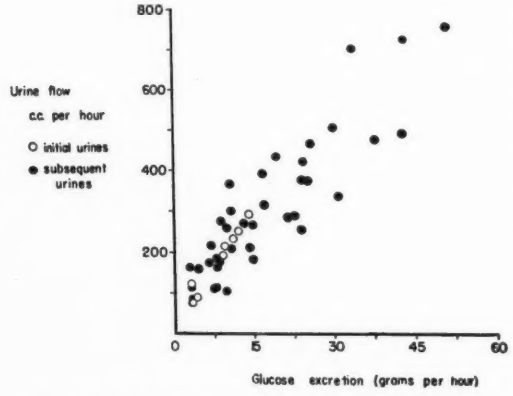

FIG. 2. The relationship of urine flow to glucose excretion in 11 patients with diabetic acidosis. The flow of urine rises in direct proportion to the excretion rate of glucose. About 21 cc. of urine are lost with each gram of glucose excreted. (After Seldin, D. W., and Tarail, R.: Metabolism of Glucose and Electrolytes in Diabetic Acidosis, J. Clin. Investigation *29*:552-565 [May] 1950.)

The extreme degree of cellular dehydration in diabetic acidosis generally is not appreciated because the low concentrations of sodium and chloride in the serum usually are construed as signs of relative water excess. Hyponatremia and hypochloremia in most instances signify a decrease in the effective osmotic pressure of the extracellular fluid and ordinarily require administration of hypertonic saline solution for correction. In diabetic acidosis, however, hyperglycemia contributes a large increment to the effective osmotic pressure, so that even low concentrations of sodium and chloride are not signs of hypotonicity, inasmuch as the effective osmotic pressure is actually elevated above normal if the osmotic effects of glucose are added to those of sodium salts. Hence, hypertonic saline solution is contraindicated in the treatment of this disorder. This is especially true when it is recognized that the oxidation of carbohydrate, by reducing the blood sugar, permits water to return to tissue cells, thus automatically resulting in a rise in concentrations of sodium and chloride in the serum.

Potassium and phosphorus metabolism change in a similar manner during diabetic acidosis.[9] Since no clinical significance can be ascribed clearly to phosphorus abnormalities, the following discussion will be confined to potassium, although the principles apply equally to phosphorus.

At the height of diabetic acidosis, tissue cells are desperately depleted of potassium, yet the serum concentration tends to be elevated. This depletion of body stores of potassium results from several processes: (1) There is a loss of potassium in vomitus, at the same time that the intake of food is negligible. (2) Cells discharge potassium at very rapid rates, as a consequence of dehydration, accelerated protein catabolism, and impaired carbohydrate utilization. (3) Urinary losses of potassium are accelerated (fig. 3, control period). Despite this cellular depletion, the concentration of serum potassium usually is elevated. This elevated concentration in the serum before treatment apparently results because of the failure of the initially accelerated loss by vomiting and renal excretion to keep pace with the effects of rapid cellular release of the ion and a contracting extracellular volume.

With treatment these processes are reversed. Extracellular volume is expanded, thereby reducing the concentration of potassium by dilution. Protein breakdown is retarded, carbohydrate utilization is restored, and potassium therefore moves back into cells. As a consequence of these processes, the concentration of

serum potassium may fall to extremely low values, leading at times to toxic cardiac, neurologic, or respiratory symptoms. Although it has been contended that the administration of exogenous glucose, by sweeping potassium into the urine,[4] plays an important role in the development of hypokalemia, the fact that potassium excretion decreases progressively during treatment notwithstanding marked increases in glycosuria (fig. 3) militates strongly against this hypothesis.

This discussion has considered diabetic acidosis thus far apart from ketosis to emphasize the impact of osmotic disturbances related to glucose. Despite the dramatic character of ketonemic acidosis, it is probable that its therapeutic implications have been overemphasized.

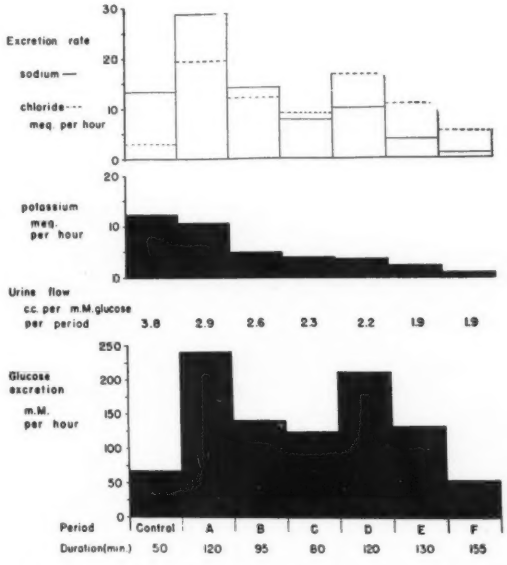

FIG. 3. The effect of repeated injections of hypertonic glucose on the excretion of electrolytes in a patient with diabetic acidosis. Severe glycosuria accelerated the excretion of both sodium and chloride. Potassium excretion is highest before treatment and falls progressively notwithstanding marked increases in glycosuria. (After Seldin, D. W., and Tarail, R.: Metabolism of Glucose and Electrolytes in Diabetic Acidosis, J. Clin. Investigation 29:552-565 [May] 1950.)

The acidosis associated with ketonemia, like the acidosis of chronic nephritis, is principally a consequence of the accession of fixed acids to the plasma which displace bicarbonate from combination with sodium. Ketonemic acidosis is unique, however, in that the major portion of the acidosis can be reversed by the restoration of the ability to burn carbohydrate: sodium ketone is then reconverted into sodium bicarbonate, and the acidosis is largely corrected. For this reason, ketone acids in the blood have been termed "potential bicarbonate." If to the low concentrations of serum bicarbonate are added the amounts of potential bicarbonate in the blood, it becomes apparent that the deficit of total bicarbonate is usually of minor proportions. The acidosis can be reversed rapidly at the onset of treatment by the administration of large amounts of sodium bicarbonate, as Guest has recently urged,[3] but this entails the danger of a late alkalosis once carbohydrate metabolism has been restored and sodium ketone burned to sodium bicarbonate. Sodium lactate is even less suitable as an alkalinizing measure, not only because of the danger of a late alkalosis, but because in addition lactate is only an alkaline salt when it can be burned to bicarbonate, a process which proceeds sluggishly in severe diabetic acidosis. During recovery, such a conversion may transpire rapidly, but at this point the need for alkali has long since vanished. Despite the profound reduction in bicarbonate, therefore, it seems doubtful that specific alkalinizing measures need be employed in the treatment of diabetic acidosis.

THERAPEUTIC IMPLICATIONS

The osmotic properties of glucose heretofore discussed have important implications for treatment. It is apparent that the marked losses of salt with glycosuria are accompanied by far greater losses of water. As a result, contraction as well as hypertonicity of the extracellular fluids ensues. The magnitude of the deficiency of water may be underestimated because the effective osmotic pressure of glucose causes a dilution of the extracellular fluid with cell water, resulting in a normal and low, instead of elevated, concentration of serum sodium and chloride. These osmotic effects of glucose, coupled with starvation, vomiting, and ketosis, eventuate in the profound disruption of electrolyte metabolism characteristic of diabetic acidosis: extreme salt depletion, hypertonicity despite low concentrations of sodium and chloride in the blood, cellular dehydration, potassium intoxication, and ketonemic acidosis.

By promoting carbohydrate utilization, and thereby suppressing ketosis, hyperglycemia, glycosuria, and cellular breakdown, insulin occupies a crucial position in the treatment of diabetic acidosis. The dosage should be especially large at the start of treatment (50 units of regular insulin per hour) when the patient is relatively refractory to its action, and subsequently reduced as carbohydrate utilization, judged especially by the fall in blood sugars, becomes accelerated. Reliance should not be placed on values of sugar in the urine as an index of the blood sugar, since the correlation is apt to be especially poor in diabetic acidosis, owing to changes in the renal tubular reabsorption of glucose, lags in urine collection, urinary dead-space errors, and the variation in urine flow which, by dilut-

ing or concentrating the urine glucose, masks the magnitude of glucose excretion.

Normal saline or Ringer's-lactate solution is required to expand extracellular volume. For the average adult patient some 3 L. should be administered within the first four to six hours after onset of diabetic acidosis. Part of this amount can be injected by clysis. Since the patient is usually stuporous, pain from clysis is no important problem; this route, by obviating a direct load on the circulation, has obvious advantages in the patient with serious heart disease.

Glucose solution, since it affords a medium for the parenteral administration of water without salt, is necessary to ameliorate hypertonicity, but it should not be given in amounts which increase hyperglycemia or glycosuria. Five per cent glucose in distilled water may be injected intravenously at a rate of about 200 cc. per hour until the utilization of carbohydrate becomes accelerated, as judged by a fall in the blood sugar level. At this time 10 per cent glucose, infused at a rate of 200 to 300 cc. per hour, seems advisable to prevent hypoglycemia. Glucose solutions should never be administered by clysis, since salt is drawn into the glucose pool at the site of injection, tending thereby to aggravate salt depletion and shock.

Finally, from four to six hours after the onset of treatment, hypokalemia tends to appear. Usually, no clinical evidences of potassium deficiency are detectable, although occasional instances of muscular weakness or frank flaccid paralysis, respiratory impairment, and myocardial failure have been reported. The best index of hypokalemia is a chemical determination of the concentration of serum potassium. Electrocardiographic signs—broadening of the Q-T interval, flattening or inversion of the T wave, depression of the S-T segment—may be helpful, although it should be recognized that such changes are nonspecific and sometimes highly misleading. If chemical and electrocardiographic determinations are not available and the patient appears to be responding and elaborating adequate amounts of urine, it is desirable nevertheless to administer potassium. This should be given in a dilute form, at a concentration not much greater than 50 milliequivalents per liter and at a rate of about 25 milliequivalents per hour. Four Gm. of potassium chloride may be added to a liter of saline solution or glucose and injected intravenously at a slow rate (over a two and one-half hour period), so as to avoid both transient hyperkalemia and severe pain along the vein. This dose may have to be repeated if nausea and vomiting prevent oral feeding. As soon as the patient is able to eat, potassium salts may be added to oral feedings, although potassium in the diet usually will be sufficient to prevent hypokalemia.

SUMMARY

Study of the effect of glucose administered in large quantities to normal human subjects is of value in understanding the distortions of electrolyte metabolism characteristic of diabetic acidosis. To counteract these disturbances of diabetic acidosis, insulin, normal saline or Ringer's-lactate solution, glucose, and potassium play important roles.

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ABSTRACT OF DISCUSSION

DR. JAMES W. SHERRILL, La Jolla, San Diego, Calif.: From the practical standpoint Dr. Seldin's work serves to reimpress us with the profound influence which hyperglycemia and dehydration play in the metabolic processes in diabetes. Hyperglycemia not only is involved in the alteration in cellular interchange in diabetic acidosis, but it also plays a role as a dehydrating agent, as so commonly is seen in uncontrolled diabetes.

The chemical processes in these experiments on normal subjects are not the same as in diabetic acidosis although the end results are essentially the same. The hyperglycemic impact is much more sudden and exaggerated than in diabetic acidosis. In diabetic acidosis the physician is dealing with insulin deficiency which blocks glucose utilization and alters cell permeability. There is the same lack of potassium in the cells and the shift of sodium from the serum into the cell. It may be reemphasized that glucose enters the cells by an enzymatic process and not by a simple process of diffusion and osmosis. It is fortunate that in diabetic acidosis this altered cell permeability to potassium, phosphorus, and sodium is easily reversible with insulin, provided glucose is not administered intravenously.

Intravenous glucose has little or no place in the management of diabetic acidosis. It simply increases the already present hyperglycemia and dehydration and prolongs the loss of much needed potassium and chloride. Fluid balance is best attained with normal saline solution and adequate insulin. My record of patients recovering from diabetic acidosis has been greatly enhanced since I adopted the rule that glucose has only one place in diabetic management, namely, in extreme emergencies of hypoglycemia which cannot be relieved by ingested carbohydrate.

From the experimental standpoint, hyperglycemia per se does not produce cellular desiccation but withdraws water

from the cells. The overall observation is correct, but it is not the direct consequence of high blood sugar levels. There must be the added factor of water loss through the intact kidney. With the newer isotope methods, Drs. Wick and Drury have determined the glucose space in totally eviscerated animals and have shown glucose concentrates as high as 1,300 mg. per 100 cc. of plasma without evidence of cellular dehydration.

DIABETES IN CHILDREN

Some Practical and Theoretical Considerations in Management

JAMES W. SHERRILL, M.D., La Jolla, California

DIABETES mellitus is serious at any age. The treacherousness of the disease is magnified in childhood. It is the poorest treated of all the chronic diseases. The role which the pediatrician accepts in guiding a diabetic child is one of grave responsibility and carries with it not only proper medical supervision but also serious consideration of the child's social, educational, marital, economic, and eugenic future.

In the treatment of diabetes the pediatrician occupies a rather unique position. By means of almost any type of diet, or no diet at all, and almost any type of insulin the child can be kept alive. Simple maintenance of life is not the goal in diabetic management, however. With the exception of coma there are few real complications. The pediatrician's position is different from that of the internist who assumes responsibility for the child after he has passed a specified but rather youthful age.

The serious nature of diabetes, especially in children, was thoroughly known in the preinsulin era. The average duration of life in the Naunyn era was from three to six months. Continuous loss of weight, weakness, and physical incapacities were the rule in adults. Once body weight was lost, it could never be regained. Diabetic women rarely conceived and, in the rare instances of pregnancy, immediate abortion was indicated to save the maternal host. The universal cause of death was coma. In the present era, death from coma is rapidly approaching zero.

The prevalence of diabetes is shown by the fact that it occupies the tenth position as the cause of death. It is found in all races and nationalities, and the incidence of the disease is the same in all walks of life and the same in rural, urban, or metropolitan areas. No information as to its etiology is available except that it is hereditary. There are no authenticated cases of diabetes as a result of trauma or infection.

From The Scripps Metabolic Clinic, La Jolla, San Diego.
Read before the Section on Pediatrics, Texas Medical Association, Annual Session, Houston, April 29, 1953.

DEFINITION OF DIABETES

If diabetes is to be treated successfully and statistical results of therapy compared and evaluated, there should be a common understanding as to what diabetes is, what controls it, and some of the fundamental principles which regulate it. The most common definition usually given to the laity is that it is a disease in which the pancreas does not produce enough insulin to burn up the sugar in the body. Unfortunately this is the definition found in practically all present-day textbooks in medical schools, and it is the not uncommon definition given by many members of the medical profession. Sugar is not specific in diabetes; if patients are treated upon the basis of such a concept, they will suffer from premature complications.

Allen[1] explained the scientific factors which regulate the disease and gave a lucid definition, namely, that "diabetes is a metabolic condition in which the individual cannot utilize the normal amount of food." Sugar is only one of the many foods. Food is measured in calories. Because of the high caloric value of fat, its restriction in the diet is equally, if not more, important than the restriction of carbohydrate. The young pediatrician will profit greatly by reviewing the vast number of illuminating scientific animal experiments which were made by Allen.

The pediatrician who was graduated since 1922 does not see diabetes in its unmodified form because its perfidious and menacing aspects are masked by insulin. Allen used dogs with partially removed pancreases in thousands of experiments during many years to demonstrate factors which regulate the disease. By feeding high caloric diets and small amounts of carbohydrate, he produced in mildly diabetic dogs severe

diabetes within a few weeks or months. High fat diets increased the rapidity with which diabetes progressed. By restricting fat and calories, he reduced severe diabetes to a mild state within a brief period. Allen established one fundamental law: namely, that there is a direct relationship between the body mass and the activity of the disease, that is, the greater the body weight or the greater the obesity, the more active the diabetes. In other words, since the diabetic patient has a permanently reduced and fixed insulin supply, the more the body size or mass increases, the less insulin is available for each pound of body weight. Reduction of body weight of diabetic dogs by low caloric diets rendered diabetes quiescent. He showed experimentally that any agent or food which increased the metabolism aggravated the disease. Thyroid extract, infections, pregnancy, and many toxic agents have such an effect. Diabetes was shown to be a disease of total metabolism so that calories from all sources, whether protein, fat, carbohydrate, or alcohol, were equally important.

Today physicians would not countenance undernutrition in any form in the diabetic program, especially in children. However, if the inerrant fundamentals of the preceding era are forgotten, the eventual results of diabetic treatment will be inferior. Although undernutrition is not now generally recommended, there is much information to show that overnutrition with free diet is responsible for the unnecessary early complications: coma, retinitis,[22] and Kimmelstiel-Wilson disease, all of which are too frequently encountered. If one examines closely the clinical and laboratory data of those who advocate a so-called "free diet," it will be noted that there is little variation in the degree of control between the control group of patients and the "free diet" group. The so-called control group is not regulated by any chemical standard or total caloric restrictions; treatment is dependent mostly upon carbohydrate restriction.

In the brief Allen era the life span of the diabetic person was more than doubled. Doctors were able to extend the life of diabetic children from three to six months or even to several years; many of these patients lived to receive insulin and are alive and well today. The era, for many of us, was a difficult one. We were in a constant dilemma; we could reduce the child's nutrition by giving a low caloric diet and extend his life or let him die promptly in diabetic coma.

DIAGNOSIS IN CHILDREN

The onset of diabetes in most children is abrupt and the diagnosis is evident. The actual increase in the metabolic rate in infants and children is reflected by profound polyuria, thirst, and weight loss, in contradistinction to more insidious symptoms in adults. Venous blood sugar levels exceeding 170 mg. per 100 cc. or capillary blood sugar levels exceeding 190 mg. postprandially, if consistent and associated with glycosuria when the child is on a general diet, are sufficient to warrant a diagnosis of diabetes. Postprandial blood sugar levels in children usually exceed 200 or 300 mg. at the onset so that the diagnosis is obvious. Postprandial blood sugar levels are as valuable and reliable as glucose tolerance tests. The latter, in most instances, give only academic information; it is seldom necessary and serves a useful purpose only in a small group of questionable cases. The urinary sugar in diabetes is glucose, which gives a sharp unquestionable reaction with simple tests. In nondiabetic melituria the reducing substances are atypical and nonspecific. No exact information is available as to just how long glycosuria and hyperglycemia may exist in children before the onset of the accepted diagnostic signs of polyuria, polydipsia, polyphagia, weight loss, and the like. In adults it is known that this period may be a number of years. In children, however, the period must be brief in view of their high metabolism. In any statistical consideration which involves the duration of diabetes and its complications, physicians, therefore, must differentiate between the actual onset of diabetes, the frank onset of cardinal symptoms, and the date of diagnosis. None of these may coincide.

At the onset diabetic children are usually overheight. This is attributed to the antagonistic or antiinsulin effect of excessive pituitary-growth hormone and is similar to the correlation of diabetes in acromegaly of the adult; practically all acromegalic adults develop diabetes. Diabetes in the obese child, especially in dystrophia adiposogenitalis, or so-called Fröhlich type of obesity, is extremely rare.

HYPERGLYCEMIA AND DEHYDRATION

In view of some of the recent recommendations for laxity in treatment and particularly the statement that it is not necessary to control glycosuria and hyperglycemia, some of the actual scientific physiologic and pathologic factors which are known to regulate diabetes might be reviewed. Physicians might also recall the responsibilities with which they are charged. They are taught and accept as a principle that any processes in the human body which deviate from normal are not only pathologic, but harmful. The whole pattern of the physician's existence is one of seeking the abnormal and striving to restore normal function to abnormal organs.

Hyperglycemia is harmful. Ricketts[19] has expressed the situation clearly, "There is an abundance of evidence to show that hyperglycemia is harmful and no existing evidence to show that it is harmless." Hyper-

glycemia is harmful because it is inseparably associated with dehydration, which has known specific effects on cellular injury. Some idea of the dehydration which exists in uncontrolled diabetes is seen in the rapid gain in weight when hyperglycemia is corrected. Gains of from 2 to 5 pounds within a few hours is not uncommon. What more evidence is needed than the experiments of Deutschmann[5, 10, 11] who showed that frogs developed cataracts when immersed in hypertonic glucose solution, provided an element of dehydration was introduced? The conclusive experiments at Yale of Rodriguez and Krehl,[20] who have recently produced cataracts in animals by hyperglycemia per se, cannot be ignored, nor can the fact that cellular alteration in the form of hydropic degenerative changes in the beta cells of the island of Langerhans are produced by hyperglycemia. The development of diabetes can be prevented in the potential diabetic patient simply by controlling hyperglycemia. Dehydration has been shown to alter the concentration of electrolytes[9] and the total osmotic pressure of the intercellular and intracellular fluids, and also to alter surface tension and adsorption of cell membranes. This alteration and loss of intracellular ions, especially potassium, probably account for the weakness and fatigability found in long-standing uncontrolled diabetes. Dehydration is known to destroy muscle protoplasm,[16] to decrease blood volume,[13] to increase blood viscosity,[17] and to decrease the volume of blood flowing through tissues by peripheral vasoconstriction.[14] All of these factors encourage anoxia in tissues.

Loss of tissue protoplasm which results from prolonged dehydration is usually accompanied by reduction in prothrombin and clotting time and is interrelated with retinal hemorrhages. Retinal hemorrhages are known to improve with control of diabetes and the use of high protein diets to restore the lost protoplasm.[21] Bird[4] demonstrated experimentally that moderate degrees of dehydration caused striking weakness in gastric wounds irrespective of the type of sutures used and also that it prevented wound healing.

One of the most interesting and instructive illustrations of the effectiveness of treatment is found when Simmond's disease intervenes in diabetes. Poulsen[18] has recently published one of 9 such instances. A woman of 30 years who for twenty years had had diabetes which never was controlled was under observation at the Steno Memorial Hospital, Copenhagen, because of diminished vision due to retinal hemorrhages. She had been admitted in coma on twenty occasions. Insulin dosage ranged from 80 to 100 units daily. Repeated funduscopic examination showed many hemorrhages and waxy exudates. Simmond's disease intervened in 1945; within a few months the patient became hypersensitive to 8 units of insulin and the urine became sugar free. With the disappearance of diabetes the vision improved, and within four years the retinal hemorrhages and waxy exudates had disappeared.

Still more conclusive scientific proof of the damage caused by hyperglycemia, which may appeal to the physician with an inquiring mind, is observed in the retina. Small retinal aneurysms, as described by Ballantyne,[3] are probably the earliest demonstrable pathologic lesions in diabetes. They are 30 to 60 microns in diameter and are just within the limits of visual acuity. These lesions are to be seen with the ophthalmoscope in 100 per cent of cases of Kimmelstiel-Wilson disease and may be seen in minor numbers after ten or fifteen years of uncontrolled diabetes. They later progress to form hemorrhages. By injection methods these miliary aneurysms are noted universally in the retinas of all diabetic patients examined post mortem. For several years, it was assumed that they were confined solely to diabetes. However, in 1951 Ashton,[2] using injection methods, noted aneurysms at autopsy in 33 per cent of the retinas of nondiabetic persons, although they were too small to be seen with the ophthalmoscope. Since they are potentially present in nondiabetic persons but do not progress even to the point of becoming visible with the ophthalmoscope, it must therefore be assumed that the hyperglycemia of uncontrolled diabetes is the sole agent which causes these aneurysms to progress to the stage of hemorrhage and visual impairment in the diabetic patient. No other factor except active diabetes could account for the progression. Retinitis must therefore be considered as a complication of diabetes and not an inescapable counterpart of the disease, as claimed by some authors.[6] On many occasions, I have seen retinal aneurysms disappear when hyperglycemia is controlled.[22]

TREATMENT

Maintaining normal nutrition and assuring proper growth and development and, at the same time, maintaining control of hyperglycemia and glycosuria are the objectives in treatment. It is not possible to maintain normoglycemia continuously throughout each twenty-four hour day, but this goal may be strived for. If one could maintain normoglycemia, it is reasonable to assume that diabetic complications would not develop. Given a patient with uncontrolled diabetes for fifteen years, some form of vascular lesion can be demonstrated. Given a diabetic patient who has had the disease thirty years and who has controlled it two-thirds of the time, that patient can be considered as having diabetes only ten years, hence no vascular disease.

To treat the disease successfully, one must exercise

great patience in the matter of teaching and instruction. Success also depends upon the intelligence of the patient, as well as his fidelity. Instruction is a matter of individual care. One of the reasons for generally poor results is the fact that diabetes extends throughout a lifetime; instruction may become monotonous because of reiteration and because the patient's self-discipline at times may pass the point of restraint. Proper management should entail no particular hardship. In reality the diet is one which the nondiabetic person should take. However, because of lack of will power many persons consume much more food than they really need.

Patients should have a complete understanding of the nature of the disease. They should be taught that treatment carries with it more profound implications than the simple restriction of carbohydrate in the diet and freedom from sugar in the urine. Since the diabetic diet is quantitative and measured in calories, each patient should be given the initial opportunity of receiving a carefully prepared diet under the supervision of competent dietitians. Within this time—which should not require a long period of institutional care, possibly a week—the physician has an opportunity to determine the degree of diabetes and something of the insulin requirement. Patients are not required to weigh their diets all of the time, but the best results have been obtained in those who refresh their memories by weighing their food periodically.

One great error in the management of diabetes in children lies in enthusiastic overfeeding at the onset of the disease. Although the onset is initiated with drastic symptoms and heavy glycosuria, diabetes responds quickly and dramatically to relatively small amounts of insulin. All diabetes is mild in the beginning. During the first weeks the disease in the child can be kept under excellent control with trivial doses of almost any kind of insulin and diet. During this time the pediatrician, in his enthusiasm, may recommend a more liberal diet and a reduction in insulin. However, this period is short-lived and tolerance is soon lost. Within a few weeks or months there is heavy glycosuria, the insulin requirement mounts rapidly, and the diet must be drastically reduced. This is an inevitably disappointing and discouraging situation for mother and child and one which can be avoided if in the beginning the diet is kept reasonably restricted and adequate insulin insisted upon. In this way as the child grows and needs arise, small food increments can be made which, in themselves, give encouragement and inspire fidelity.

When a child develops diabetes, naturally there is great anxiety in the family. One of the physician's first objectives is to give reassurance that diabetes can be controlled and that plans for the child's future should not be altered. The child should make plans for the same education; he should attend the same social functions; he should be delegated the same home duties and responsibilities; and he should be disciplined in the same manner as the nondiabetic child. Parents should be instructed not to discuss diabetes, food, or insulin at the family table and reminded that the course of family life does not revolve around a urine test.

There is no way in which to calculate the diet of a diabetic child on the basis of height, weight, age, or any other one factor. The control of diabetes depends upon many factors—age, sex, height, weight, duration of the disease, degree of previous control, athletic habits, and especially the state of nutrition. Food tables and food requirements of the normal child are not applicable to the diabetic child, and therein is found one of the universal causes of failure. Since diabetes is a condition in which the child cannot utilize the normal amount of food, attention should be directed to a diet which meets his metabolic needs rather than to one meeting the theoretical needs of the normal child. The standard food tables for normal children are excessive for the diabetic child because a great number of extra calories have been added empirically. Diabetic patients do not suffer on maintenance diets. It may be recalled that many obese nondiabetic patients cannot lose weight on diets of 1,000 calories, further evidence that so-called normal tables are unduly high. Many nondiabetic women engaged in reasonable activity maintain good nutrition on from 1,400 to 1,600 calories. I have not observed deficiency in growth, development, or nutrition, nor have I noted the development of any psychologic insults in children with moderate caloric restrictions. Neither have I observed the Kimmelstiel-Wilson syndrome in diabetic children who have been faithful to their programs even into the third and fourth decades. Kimmelstiel-Wilson disease is confined to that group of patients who have followed a so-called free dietary program, but that is the responsibility of those who recommend it.

In the diet protein deserves the first consideration. It is necessary for strength, growth, and the prevention of hunger and weakness. Protein is the most valuable of all the foods, especially when its relatively low caloric value is considered. The average diabetic child should receive 80 to 100 Gm. daily with proportionately large amounts after the age of 12 years. The carbohydrate allotment should range from 160 to 200 Gm., which is ample for a good supply of fruits and vegetables, insuring a normal vitamin allotment. The carbohydrate allowance should rarely exceed 190 Gm. Patients should receive a fairly large allotment of

starch and should be instructed to distribute it equally between the three meals. The only specificity which carbohydrate has in diabetes is the so-called law of diminishing return: the greater the percentage of carbohydrate taken, the less is utilized. It is better to confine carbohydrate calories to the lower percentage foods and use them rather than to take the higher percentage foods and lose them. Fat is the least important of all the foods. It should be adjusted according to the child's weight and growth. Allotments should range from 70 to 100 Gm. with a general average of 85 Gm. Fat can be raised to very high levels for a considerable period before glycosuria occurs, but, once it does, diabetes is severe. The disastrous result of a high fat diet is now a matter of history.

The distribution of the food elements in the daily meals should simulate the normal. Many children are satisfied with equally divided meals. The distribution of one-fifth, two-fifths, and two-fifths for breakfast, dinner, and supper, respectively, is satisfactory. Small midmeal feedings, with a minor amount of food on retiring, is frequently helpful. Any program of food distribution which deviates to any extent from the normal will work a hardship on the mother and will not invoke control.

Insulin

Insulin has been in general use for thirty years and still very little is known about its action. The fact that it has an over-all effect on protein,[15] fat,[7] and carbohydrate,[23] however, substantiates previous knowledge that diabetes is not simply a matter of carbohydrate metabolism but one in which all foods are involved. The site of action of insulin is unknown. Recent evidence indicates that insulin accelerates the transfer of substances from the blood stream to the cells. The mechanism is not merely one of diffusion but rather the action of insulin on an enzyme system, probably a phosphorylation type of reaction.[8]

Insulin for the diabetic child should be started as soon as the diagnosis is made. Prolonged delay simply encourages nutritional and growth deficiencies.

One of the most common reasons for failure in treatment is the attempt to control advanced cases with one dose of insulin. This impossibility should be readily admitted. Very few cases, except early mild ones, are controlled well with one dose of any type of insulin. The mode of action and duration of the various insulins are well known. In moderate or severe cases it is known that one type of insulin, when increased to any extent, causes wide oscillations of the blood sugar from hyperglycemia to hypoglycemia. There is no simple or short-cut method to good control. The best control is obtained by using quick-acting crystalline and protamine zinc insulin in separate syringes not less than one-half hour before breakfast. The quick-acting insulin allays hyperglycemia induced by breakfast and is effective until past midmorning. By this time the protamine zinc insulin has become effective and continues so until the early morning hours. The ratio of protamine zinc insulin to crystalline insulin should be about 3:1 or 3:2.

Good control can be maintained on an adequate diet with 25 to 45 units of insulin daily. When doses of 75 to 100 units are encountered, it is reasonable to believe that the child is obtaining food from outside sources or his assigned diet is far in excess of his requirements. There is good evidence that the actual insulin deficiency in diabetic patients does not exceed 40 units daily. Although patients may remain aglycosuric with excessive amounts of food and tremendous amounts of insulin, this is no criterion by which to judge good diabetic treatment. The diabetic patient cannot use a large number of calories, so that with such artificial depression of the blood sugar level by giving excessive amounts of insulin, early complications in the disease may be expected.

Experience with diabetic children has taught me that control by chemical standards offers the best and safest method for guidance. Experience has taught me one valuable lesson, namely, the child's safety is sacrificed if his feeling of well being or general appearance is used as a guide. With trivial amounts of insulin children may feel and look well until they suddenly go into coma. The outward appearance of an overweight and robust child is no indication of the variance of chemical components of blood metabolites. Children should be muscular and strong but not overnourished.

One other valuable factor for the child's safety is a careful explanation to the parent of the importance of an adequate amount of insulin. Since the blood sugar level may rise to a considerable degree before sugar is excreted in the urine, inadequate amounts of insulin will mean constant hyperglycemia. It is better to instruct the parent to see how much insulin the child can take, obviously avoiding a reaction, than it is to see how little he can take and barely avoid glycosuria. The parent should understand that minor doses are dangerous and later have to be overcompensated. My experience coincides with that of Jackson,[12] who has shown that reactions are fewer in the child with controlled diabetes than in the one with uncontrolled disease, a fact easily explained on the basis of the mechanism involved in reactions. The occurrence of reactions depends not only upon the low point to which blood sugar levels fall but also on the rapidity and the precipitous heights from which they fall.

SUMMARY

There is sufficient preexisting information concerning the factors which regulate diabetes to encourage one to maintain good control of the disease; some of these are control of obesity, a regulated diet of adequate calories sufficient for nutrition and growth but avoidance of a high caloric diet, and chemical control of hyperglycemia and glycosuria. There is no scientific evidence that hyperglycemia is harmless but considerable evidence to show that it is harmful.

Adequate control is not determined solely by the child's appearance or his apparent well-being, for these may be misleading. One common error in the management of diabetes is the use of too little insulin or the attempt to control diabetes of any degree or duration with one dose of any type of insulin.

This report is based on thirty-five years' experience with 400 children who developed diabetes at 15 years or younger.

Kimmelstiel-Wilson disease is confined to the group of patients who follow a so-called "free diet."

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ABSTRACT OF DISCUSSION

DR. DONALD W. SELDIN, Dallas: Dr. Sherrill has vigorously emphasized some of the deleterious consequences of hyperglycemia. He has pointed out how high blood sugar levels lead to glycosuria, which sweeps large amounts of salt and water into the urine. In addition, the hyperglycemia draws water out of body cells producing severe cellular desiccation. These harmful osmotic effects of glucose may be prevented by judicious dietary control together with insulin administration.

It should be emphasized, however, that hyperglycemia sufficient to induce significant disturbances in salt and water metabolism must usually be fairly severe. If one attempts to maintain the blood sugar continuously within the normal or near normal range, the danger of inducing hypoglycemic attacks mounts greatly. It is not a mark of successful treatment to purchase a normal blood sugar at the price of recurrent hypoglycemic episodes. It often seems wise, therefore, to permit a minimal and asymptomatic glycosuria in severe diabetes, thereby minimizing the dangers of insulin reactions.

Some investigators have argued that hyperglycemia may lead to the development of profound degenerative complications, the most serious of which is Kimmelstiel-Wilson's disease. This point can not be definitively settled at present. There is reason to believe, especially from the studies reported from Scandinavian clinics recently, that this complication is more closely linked with the duration of diabetes than with the control of hyperglycemia and glycosuria.

No one advocates license in the treatment of diabetes. On the other hand, rigid control of diabetes which achieves a sugar-free urine at the price of intermittent hypoglycemia seems equally undesirable. This is especially true if, to control glycosuria completely, rigid restrictions are placed on the patient's activities and behavior which may lead him to feel, be he child or adult, that he is socially stigmatized. Profound emotional disorders, perhaps as serious as diabetes itself, may eventuate under such circumstances. A reasonable goal would therefore seem to be reducing hyperglycemia and glycosuria to the minimum amount commensurate with avoidance of insulin reactions, at the same time permitting the patient a sufficiently flexible regimen to facilitate as normal a pattern of behavior as is possible.

Essay Contest on Sterility

The most outstanding essay on the subject of infertility and sterility presented to the American Society for the Study of Sterility by the March 1 contest deadline will be a part of the program of the group's 1954 meeting, and the winner will receive a cash award of $1,000. Information about the contest may be obtained from the society's secretary, Dr. Herbert H. Thomas, 920 South Nineteenth Street, Birmingham.

Medical periodicals published in Texas prior to 1900 are desired for the Library of the Texas Medical Association. Information about such publications should be sent to the Library, 1801 Lamar Boulevard, Austin.

Experiences with a Vitallium Intramedullary Hip Prosthesis

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THE following report is of the experiences encountered with 25 consecutive patients in whom an intramedullary type of Vitallium prosthesis was inserted to relieve a painful hip condition. The first of these patients was operated on February 20, 1951; the experiences cover slightly more than a two year period. In the beginning, the hip prosthesis was used only as a salvage procedure in cases having either instability or severe pain, such as in nonunion of the fractured neck of the femur or in aseptic necrosis of the head. Later it was used for primary arthroplasties. During the early period a change was made in the style of the prosthesis model. The major concern at the time was not only in evolving an operative technique that would be swift, simple, and sure, but in evolving a prosthesis that would provide stability and relieve pain and which would have no harmful after-effects on the host in whom it was placed. My early tribulations have helped me to evolve a satisfactory technique, and the two years' experience has given me some definite ideas as to the behavior of the prosthesis in the human body.

CRITERIA FOR PROSTHESIS

In a preliminary report,[1] ten criteria for an ideal prosthesis were outlined. It still is believed that, with one exception, all ten are necessary. The exception is the feature of anteversion, which is not believed to be essential to the good functioning of a prosthesis. Although anteversion exists in the vast majority of human skeletons, it occasionally is absent. Hips without anteversion seem to function as well as those possessing it. This is fortunate, since, from the manufacturers' standpoint, anteversion incorporated in a prosthesis means that one must have a right and left model and not a universal one that can be used for either hip. The present prosthesis no longer contains this anteversion, and it seems to function as well as those which previously possessed it. Aside from this one change, the ideal prosthesis still should fulfil ten criteria:

Head Size.—The prosthetic head should be as nearly the same size as the acetabulum as possible. The prosthesis is made in three sizes: large (1⅞ inches in diameter); medium (1¾ inches in diameter); and small (1⅝ inches in diameter). One should use the largest head size consistent with firm socketing.

Anatomic Shape.—The shape of the head and neck of the prosthesis should be anatomically similar to that of the normal femur. A central fovea in the head is avoided, since there is no ligamentum teres attached to it. Anteversion is not believed essential.

Nonelectrolytic Metal.—The material used for the prosthetic head should be absolutely nonelectrolytic. Vitallium is the material of choice. Although at present more expensive than those artificial hips made of acrylic, nylon, or steel, it better withstands wear and tear, and one can always be assured that a reaction within the body will never occur due to the material itself. This assurance is not present when using steel.

Sterility.—The prosthesis should be capable of being sterilized by autoclave methods before being inserted. Since infection is the greatest single factor nullifying the beneficial effects of the operation, pressure heat sterilization always should be insisted upon. Even prolonged periods of soaking the prosthesis in antiseptics is no guarantee of sterility.

Easy Insertion.—Since most of this surgery is done on the aged patient, the prosthesis must be easy to insert in order to minimize the danger of shock.

Anterior Incision.—Although some surgeons prefer the posterior Gibson approach to the hip joint, I still believe the prosthesis is inserted best through an anterior incision, by which division of any muscle can be avoided. The shape and design of the prosthesis favor the anterior approach for easier insertion. The incision allows for early postoperative ambulation without the necessity of prolonged bed rest or plaster fixation.

Adequate Head and Neck Length.—It is important that the prosthetic head be so fashioned that when it is correctly inserted the total length from the dome of the head to the outer aspect of the greater trochanter is adequate enough to allow the gluteus medius lever mechanism to function properly. If this length is too short, several unfavorable results will develop: A lurch will occur as the patient walks. The tension of the muscles will not keep the head in the acetabular socket. Dislocation may occur, as in some instances where a Judet prosthesis has been used in the presence of an absorbed (shortened) neck of the femur. Therefore, the length of the neck of the three prostheses has been increased with the head size. Furthermore, the overall length is taken into consideration in the individual case during surgical resection of the head, and the length of neck resection is varied to maintain

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proper distance. From practical experience this works out well. None of the patients in whom there was not dislocation postoperatively has developed lurch.

One-Piece Appliance.—It is important that the prosthesis be composed of one piece of metal. Whenever two or more pieces of metal are jointed within the body, they may come apart or the metal break at the junction point. Metal fatigue stem fracture is not seen soon after operation, but breakage may occur months or years later. Every effort must be made to avoid it. It is impossible to make a prosthesis as thick in one plane as it is in another, particularly at the junction of the neck and the collar of the prosthesis. Yet it is at this very point that metal fatigue may occur.

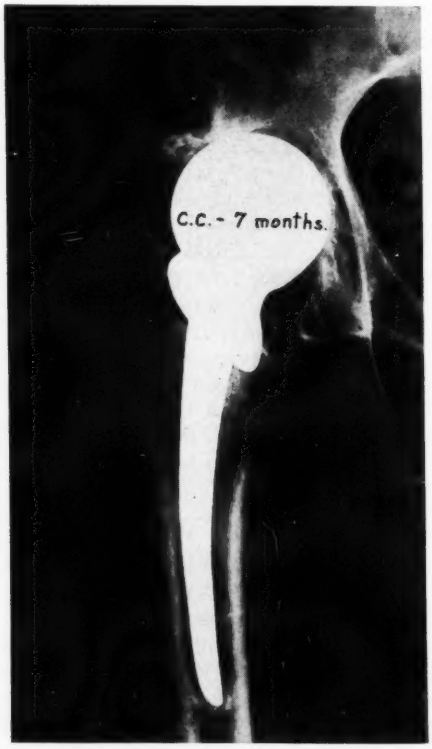

FIG. 1. A roentgenogram taken seven months following arthroplasty shows finger-like bone density curling around the lower tip of the prosthetic stem, immobilizing the prosthesis. Absence of any cradle about the upper half of the stem indicates reparative bone growing firmly about the prosthesis.

Coxavara.—The shape of the prosthesis with the long stem extending down the shaft gives assurance by the engineering principle employed that the prosthesis will not angulate into coxavara unless the shaft fractures.

Adherence of Bone to Prosthesis.—A final and important criterion for the ideal prosthesis is for its shape to encourage the growth of reparative bone to form about it to such an extent that it becomes similar to a steel girder around which concrete has been poured. The prosthesis should become incorporated into the bone and give the bone added strength. The prosthesis when introduced into the medullary cavity is not always secure and tight. This is because the diameter of the medullary cavity tends to be larger in older people and smaller in younger age groups. In some elderly people, the fit is loose and some toggle motion takes place between the prosthetic stem and the side walls of the shaft. As bone repair takes place, this toggle motion diminishes until, as a rule, by the end of the fourth month it ceases. Often a finger-like projection forms from the lateral femoral shaft wall, curling around the distal tip of the prosthetic stem and anchoring it firmly in place (fig. 1). This can be seen as early as the third month in the roentgenogram. In one instance on personal observation and in two instances reported by confreres, the prosthesis, after six months, has been found firmly anchored to the bone, so it could not be tapped back out the shaft. This does not mean that all prostheses become firmly enclosed by bone, but it does mean that the design of the prosthesis tends to allow this to occur.

OPERATIVE TECHNIQUE

The usual Smith-Petersen approach is employed in the operative technique. The interval between the sartorius muscle and the tensor fascia is sought. Care is taken to preserve the lateral cutaneous nerve of the thigh in the upper inch of the incision, since its division may cause a painful neuroma. It is frequently necessary to strip off part of the origin of the tensor fascia from the anterolateral crest of the ilium in order to give enough room comfortably to insert the prosthesis later in the operation. This muscle origin is always replaced, and it is the only muscle origin disturbed during the operation. Next is sought the interval between the rectus muscle and the tensor fascia, leading the operator to the ascending branches of the lateral femoral circumflex artery. These are isolated, clamped, and ligated. These arterial branches supply nourishment to the capsule and part of the neck of the femur. Since the neck is to be removed at its base, one need not be worried about its essential blood supply. No ill effects have occurred from the ligation of these vessels.

The capsule of the hip joint is then exposed by retracting the rectus muscle medially, being careful to preserve the oblique head of origin of the rectus. The capsule is not opened in the usual anterior fashion but is opened along its superior border by an incision which runs laterally and distally from the acetabular

rim toward the greater trochanter and then turns sharply medially toward the lesser trochanter. This gives a capsular flap which can be retracted medially. The purpose of this flap is to give a firm anterior support to the prosthetic head when it has been placed in the acetabulum. The flap will prevent subsequent dislocation, since dislocation usually occurs by the leg externally rotating and the head falling out of the acetabulum anteriorly rather than superiorly. In those cases of primary arthroplasty where the capsule needs to be excised, this supporting anterior flap, of course, cannot be utilized. In the usual case of nonunion, the capsule is saved as a flap and is not removed as has been advised by some authors for the temporary relief of pain. One dislocation and one subluxation occurred in the series of cases under discussion after an anterior incision was used. Closure of an anterior capsular incision is not always strong enough to hold the prosthetic head firmly in the acetabulum. There has been no instance of subluxation or dislocation when the superior capsular incision has been employed.

In cases of nonunion, it must be determined at this point in the operative procedure whether one is going to make up for the shortening present by capsular division or accept the shortening present and simply place the prosthesis at a lower shaft level. In those cases where no shortening exists, the nonunion site between neck and head is separated by the periosteal elevator and the dead femoral head removed. I believe it better practice to split the head into two pieces with an osteotome than to risk scratching the articular acetabular cartilage by the vigorous use of a spoon or similar instrument wedged into the joint. Similarly, in cases of aseptic necrosis, it is better to divide the neck of the femur with an osteotome and to split the head into two or more pieces than it is to dislocate the joint forcibly and run the risk of possible fracture of the femoral shaft. In those cases of malum coxae senilis in which a large deformed head is present, one should never attempt to dislocate the head from the socket, since it is quicker and less traumatic to guillotine the femoral neck and split the head into two or more pieces for its removal. After the head is removed, further trimming of the femoral neck is done with either the motor saw, osteotome, or rongeur. The line of this cut base of neck lies about 30 or 40 degrees from the center line of the femoral shaft, and its medial edge passes through the calcar femorale slightly above the lesser trochanteric level. If added length is required to make up leg shortening, the capsule at this point is stripped off medially and posteriorly from the remainder of the femoral shaft. This provides a long tube or capsular envelope into which the prosthesis later can be inserted. The capsule should not be divided from the acetabular margin to regain this length, since complications will ensue in the subsequent months, and one runs the danger of either dislocation or subluxation.

The medullary soft, cancellous bone is scraped out of the base of the neck, using a curette. Usually no special instrument is required to do this. Some, however, prefer to bend a curette about 1 cm. from the tip in order to have the cup of the curette pass down the shaft close to the calcar. Since one only needs to remove about 1 cm. in depth of this cancellous bone before the examining finger can push the remainder before it, this should not be a time-consuming procedure. The prosthetic stem is then introduced into the opening of the bone and passed distally down the shaft. Care must be taken that the stem does not pass out through an old Smith-Petersen nail hole present in those cases of nonunion previously nailed. This minor trap is easy to fall into, but it presents no problem if one knows of it and seeks to avoid it. The first three-quarters of the stem will pass easily into the medullary cavity of the bone. This is facilitated by rocking the prosthesis from side to side as it is being inserted.

The prosthesis should not be driven home with an impactor except for its last .5 cm., since there is danger, due either to improper insertion or to the bone being soft and decalcified, of having this force fracture the shaft. In elderly people the diameter of the shaft is larger and the fit usually is loose. In younger people the fit is more snug and the prosthesis may be tapped home for that last .5 cm. to obtain a firm, secure fit. The collar of the prosthesis should settle down on the base of the neck and should be well seated in all of its circumference. The calcar femorale receives the angle of the stem and the collar of the prosthesis.

Reduction is accomplished with the leg held in marked external rotation and with traction applied. The assistant attempts to pull the leg distally while the operator pushes on the prosthetic head from above. Under no condition should the leg be internally rotated with force to secure this reduction. There is danger of fracturing the femoral shaft by vigorous internal rotation. If with this traction distally, combined with a push on the prosthesis from above, the head does not descend to the proper level of the acetabulum, more base of the neck and calcar need to be removed until the prosthetic head comes down to the proper level. The use of a skid in forcing the head into the acetabulum is dangerous. The head should be reduced without undue tension. As the prosthetic head moves into the acetabular socket, the leg may be internally rotated to its normal position. The hip then usually is stable, and full flexion, abduction,

and rotary movements should be possible without dislocating the head.

The capsular incision is closed with interrupted chromic sutures. This closure should be firm and tight. The remaining muscles fall together and need no particular deep fascial sutures. The tensor fascia muscle, if stripped off from its origin, is sutured firmly back into place. The upper thigh and joint are bound with a compression spica flannel bandage to prevent hematoma. Although some surgeons prefer the use of a plaster boot to hold the foot in abduction and internal rotation, I do not consider this necessary as long as the proper capsular incision has been made. It is anticipated that motion will be begun immediately, and in some instances the patient is allowed to walk with crutches the following day. Common sense, of course, must be employed in allowing an aged patient to walk on a decalcified femoral shaft after many months of confinement to bed.

INDICATIONS FOR PROSTHESIS

The primary indication for the use of this prosthesis is in the aged patient with nonunion of a femoral neck fracture in which absorption of the neck has taken place. The simplicity of the operative procedure justifies its use in the aged patients who will not withstand the shock of much surgery.

A second indication for use of the prosthesis is in those conditions in which painful hips due to aseptic necrosis of the femoral head following union of a fractured neck of femur still retain a good acetabular socket with minimal arthritis visible in the roentgenograms. Such patients are seen infrequently. Most patients with an aseptic necrosis do not show a good acetabular socket without thinning of the cartilaginous joint space. There is usually an associated alteration of the acetabular socket conforming with the deforming effect of the femoral head. In my experience, these patients need some type of arthroplastic reaming out of the acetabulum in addition to the insertion of a prosthesis if subsequent pain is to be avoided.

The third indication for the use of this prosthesis lies in that group of patients with severe arthritis in both hips in which an arthrodesis of one hip is contemplated and some type of reconstructive procedure is needed for the other hip.

The primary use of prosthesis is fresh fractured necks of the femur in the aged is controversial. Although some surgeons believe it has a place in the very aged who have high fractures of the femoral head close to the head itself, I still believe that the procedure of choice is a Smith-Petersen nailing rather than the use of this appliance. A satisfactorily healed fracture following a successful nailing still gives a better result than that found in most hips in which a prosthesis has been employed.

Use of the prosthesis as a primary arthroplasty for painful, degenerative arthritis of the hip gives one food for thought and speculation. The indications for such a primary arthroplasty are essentially the same as those for use of the Smith-Petersen cup. My background leads me to believe that a hip fusion is the operation of choice for a painful monoarticular hip lesion, particularly in the case of the young wage earner who works long hours. Where arthroplasties are indicated, however, this prosthesis may be used. Five arthroplasties have been attempted by this method. The simplicity of the operative procedure and the rapid recovery of function without the necessity of elaborate rehabilitation apparatus and exercises is striking. The results in this small group have been superior to other types of arthroplasties I have performed.

COMPLICATIONS

In the 25 cases reported herein, there have been no deaths as a result of operation. There has been 1 acute dislocation with infection. There has been no breakage of the prosthesis.

The most discouraging complication is that of infection. For this reason it is reemphasized that complete autoclave sterility of a hip prosthesis should be employed. Danger of lighting up a latent infection always is present in reoperating in a field where surgery has been done before. It is believed wise, therefore, to extract the Smith-Petersen nail, when present, several weeks prior to the operation for insertion of the prosthesis. This rule is not rigidly adhered to. When infection does occur, the prosthesis, of course, must be removed. The simplest redemption procedure is then to cut loose the gluteus muscle attachment from the greater trochanter and place the trochanteric region of the femur into the acetabulum with the leg in abduction and encase the body in a hip spica. This Colonna type of procedure is necessary for subsequent stability of the hip when it is anticipated that the patient may ultimately walk. It is a mistake simply to remove the prosthesis and leave the hip free to ride above the acetabular level in an adducted, externally rotated hip position which leaves a worthless lower limb. These hips should be treated, therefore, in much the same fashion as an infection occurring after a Smith-Petersen nailing of a fractured neck of femur.

The second most dangerous complication is fracturing the femoral shaft, either while inserting the prosthesis down the shaft or while attempting to reduce the inserted prosthesis into the acetabular socket. This danger cannot be stressed too strongly. Many

femoral shafts are decalcified and will break easily. The prosthesis should be inserted gently and should not be driven home down the femoral shaft. Serious shattering occurred in 1 case in my series, and a primary fusion of the hip joint was done. A minor fracture occurred in another, but healed successfully with the prosthesis left in situ. It should be stressed again that when it is time to reduce the inserted prosthesis into the acetabulum, this should be done with the leg held by traction in gentle external rotation. The operator should push distally on the prosthetic head from above. Internal rotation should not be done until the prosthetic head is socketed in the acetabulum.

A minor common complication is inserting the prosthesis incorrectly so that its stem passes out through the old Smith-Petersen nail track instead of going down into the medullary cavity of the shaft of the femur. This is surprisingly easy to do. A fixed rule of taking roentgenograms at the completion of operation with the patient on the table will insure that a proper seating of the prosthesis is present.

Excision of the anterior (or total) capsule has been advised by certain authors. Some surgeons have experienced early dislocation of the prosthesis following this procedure. While this excision of the capsule may be necessary in some arthroplasties for arthritic hips, such cases should be immobilized in abduction-internal-rotation plaster boots until soft tissue healing is completed. If the socketing of the head has been deep and secure, these boots are not necessary.

Subluxation occurred in 1 case in this series, five and one-half months after operation. The hip had seemed, in check-up roentgenograms, to be well socketed for the first four months, and the patient had been actively bearing weight and walking about. The capsule in this patient had been cut loose from the acetabulum. The psoas tendon also had been divided in order to gain 1½ inches in length to offset the shortening resulting from overriding of a nonunion fractured neck of the femur. At reoperation a mass of scar tissue was found medially; it completely filled the acetabulum and prevented reduction of the prosthesis into the socket until it was excised. A shelf stabilization was necessary in this case to insure stability. Subsequent attempts to gain length have been achieved by stripping loose the capsular insertion from the femur and being careful not to cut the capsule loose from the acetabular rim. No subluxations have occurred using this technique in later cases.

RESULTS

This prosthetic operative procedure has been done for but a little more than two years, in a mere 25 cases. The results must be viewed from several standpoints. Since its primary use has been as a salvage procedure in elderly patients, one must not except such a high standard of results as one would expect if the prosthesis were used in a younger age group in whom a primary arthroplasty is contemplated. Considerable information has been gained from the experience with even this small series, as well as from the experience of confreres who have used the prosthesis in an equal number of cases, yet elaborate claims are not wise. It should be stressed again that, in my opinion, a satisfactorily nailed and healed fractured neck of the femur is superior to most cases in which a prosthesis has been used. Surgeons planning to use this prosthesis, however, naturally wish to know what can be expected of it from the standpoint of motion, limp, muscle power, limb control, and relief from pain. Too, they wish to have some knowledge of how the body tissues tolerate such a large mobile piece of metal and of what can be expected in the future. The following results have been noted:

Hip Motion.—The motion of the hip, following the insertion of the prosthesis, depends upon whether the prosthesis was inserted after simple removal of an un-united fractured head, whether extensive capsular division was done to gain length, or whether it was necessary to do an arthroplasty on the acetabular side of the hip joint. In the first category, where a nonunion has existed for but a few months with no shortening and only the dead head needs to be removed and the prosthesis inserted, motion has been excellent. These hips seem to have essentially as normal a range of motion as the unaffected hip. Flexion, internal rotation, abduction, and control of the limb have been almost normal. On the other hand, in those hips in which extensive capsular division was necessary to gain length because of overriding and shortening, motion has not been as complete as in the first group. Here flexion has usually been past 90 degrees. There has been some limitation of internal rotation and abduction. Persons with such limitations are able to sit normally and to handle their legs without stiffness being a major concern. After an arthroplasty has been done, either because of degenerated acetabular cartilage following aseptic necrosis or because of a primary degenerative lesion, motion has been somewhat limited. However, it has been more than that of the group in which extensive capsular division was necessary. In other words, in those cases falling into the second or third category motion has been satisfactory from the patient's standpoint, although the surgeon might have hoped for a more complete range.

This discussion of range of motion naturally excludes those cases which were total failures. Four failures occurred. It would not be sensible to average into the successful cases a case of total failure such as one in which the shaft was fractured and a hip

fusion then done; nor to average in the range of motion of another case in which dislocation and infection were present and the prosthesis was removed. Nor would it be sensible to include in the average a case of aseptic necrosis which was so painful on examination that a true range of motion could not be determined. It is believed that this exclusion of the total failures is justifiable when one wishes to determine what motion is to be expected when the case is uncomplicated.

Muscle Power.—In no case has the muscle power of abduction or of other motions returned to normal. Despite this, the patients with successful results do not have an abductor lurch. This shows that the power of the gluteus medius mechanism is sufficient for normal use. Stability is, of course, present in these patients. Although most patients in the successful group are able to climb stairs using the prosthetic limb to sustain body weight in both ascending and descending, the examining surgeon does not find full muscle power.

Pain.—Deleting the 2 cases of infection, 1 case of a fractured femoral shaft in which arthrodesis was done, and a fourth case in which a slow subluxation occurred after the fifth month, one is left with an evaluation obtained in 21 remaining cases. Two of these remaining 21 patients definitely were not sufficiently relieved of their pain to label the operation successful. They both had aseptic necrosis of the head of the femur in which associated acetabular arthritis had developed. The prosthesis was inserted without doing a primary arthroplasty of the acetabulum (reaming out the degenerated friable articular cartilage to form a new socket). The failure to obtain relief of pain in these 2 cases is thought to be due to the insufficiency of the arthroplastic procedure, since other patients in whom the articular cartilage was reamed out have obtained satisfactory, painless hips.

The decision to do a complete arthroplasty (reaming of the socket) is important. To perform it is more shocking to the patient and more time-consuming, and there is a greater loss of blood. Many of these sockets present cartilage that is not thick, glistening, and normal. At what point a fibrillar, soft, degenerated cartilage should be considered dangerous to leave in situ is not known. As a general rule, it is believed that if one is in doubt, the cartilage should be removed.

The remaining 19 patients have been satisfied with the relief of pain they obtained. Although 3 of this number occasionally complain of pain, they are definite in their statements that the major portion of their pain has been relieved. This leaves a remainder of 16 patients who have complete relief of their preoperative pain.

Canes and Crutches.—To date, only 5 patients are able to walk without crutches or a cane. This has been a discouraging factor to the surgeon but apparently does not represent a disappointment of great moment to the patient. Most of these patients seem capable of walking across the room without the use of a cane, but state that they do not feel secure without it and still wish to use a support as an everyday practice of safety. This may be due to the aged group in which the surgery has been performed. A similar state of affairs has been seen in those aged persons with healed fractured necks of the femur. Since the strength of these hips seems to improve well into the second year following operation, it can be expected that more patients will be able to walk without canes and crutches at a later date, a behavior similar to that reported following Vitallium cup arthroplasty.

Body Toleration to Prosthesis.—During the two-year period the prosthesis has been observed, the following body reactions have been hinted at.

FIG. 2. A roentgenogram taken one year after arthroplasty depicts increased density of the calcar at the junction of the stem and collar of the prosthesis and thickening of the shaft around the distal third of the stem. Joint space is still thin. The prosthetic model is of the earlier type. (After Thompson, F. R.: Vitallium Intramedullary Hip Prosthesis: Preliminary Report, New York State J. Med. 52:3011-3020 [Dec. 15] 1952.)

1. *Acetabulum:* What happens to the acetabulum after the prosthesis has been in place? In those cases in which a wide joint space was present at the time of the removal of the nonunion of the fractured neck, the joint space has retained its width and the cartilage has well tolerated the Vitallium metal prosthesis. In the more numerous cases in which there was some thin-

ning of the joint space, there has been noted in the follow-up roentgenograms a tendency to bone condensation or sclerosis in the acetabulum (fig. 2). This is at the point of maximum stress transmission through the acetabular roof (fig. 1). In 2 cases in which an arthritic joint was present and the acetabular cartilage did not have an arthroplasty, this condensation appeared rapidly and is believed to be responsible for part of the pain resulting. There has certainly been no tendency for a slightly damaged, worn-out joint cartilage to revert back to normal following the insertion of the prosthesis. Those patients having an arthroplasty or reaming out of the cartilage have shown the usual thin joint space that is seen following any arthroplasty. There has been no particular tendency to form the sclerotic area of bone condensation at the point of maximal weight stress. It is anticipated, however, that all such arthroplasties may not be free of change in the future. No case to date has shown an acetabular protrusion of the prosthesis into the pelvis. It was expected in the beginning that this would be a definite complication, since it has been known to occur with Vitallium cup arthroplasty. Its occurrence is still anticipated.

FIG. 3. A roentgenogram taken fourteen months after the prosthesis was inserted for nonunion of the fractured neck of the femur shows thickening and increased density of the calcar.

As noted previously, there has been one slow subluxation of the prosthesis from the acetabulum after it had been well socketed for five months. This occurred in a case in which extensive capsular division had been performed to gain length, the capsule having been cut loose from the rim of the acetabulum and the psoas tendon divided. The subsequent healing of scar tissue seemed to exert an outward pressure on the prosthesis, causing subluxation. The capsule evidently had not reattached to the acetabulum. This case was revised by performing a complete arthroplasty and inserting a shelf to stabilize the head. Although limited motion resulted, the hip thereafter was stable. From this experience I advise that no capsule be opened anteriorly nor removed from the rim of the acetabulum.

2. *Femoral Neck:* Some cases have had a small portion of the base of the femoral neck left, and the collar has been allowed to rest on this neck. The neck nubbin has absorbed in the subsequent months following operation, until the collar settled down well over the calcar and formed a broad base of contact extending from the greater trochanter along the intertrochanteric line toward the base of the lesser trochanter. In other words, if a portion of the base of the neck is left, it tends to absorb down to this basic line or junction between the shaft and the femoral neck. In some patients in whom there has been a generalized demineralization of the bone as a result of the nonunion, there has been absorption of a soft calcar femorale. This absorption has not tended to be greater than 5 mm., and the absorption seems to stop when the density and strength of the bone increases with active use. In severely decalcified femoral shafts, it is possible that more absorption will take place. For this reason, it is wise to insist upon the use of crutches for a period of time dependent on satisfactory bone density as revealed by roentgenogram. When the hip feels secure to the patient, more weight will automatically be borne. The urge to rush the resumption of weight-bearing is a good index of nature's ability to tolerate the strain of the prosthesis on the bone.

There has been an occasional overproduction of calcium around the collar near the junction of the calcar. This appears to be more a calcification in the soft tissues in the neighborhood of the prosthetic collar which partially overlaps and curls about the collar. This seems to have no ill effect upon the function of the prosthesis nor upon hip motion.

3. *Femoral Shaft:* After the prosthesis has been in place for months there is first noticed in the femoral shaft a condensation of bone in the region of the calcar where the junction of the prosthetic stem and collar meet to bear the major portion of the body weight on the bone (fig. 3). There is a further thickening of the calcar in this area and the increased calcium density may extend down the medial cortical wall for a distance of more than 1½ cm. This is believed to be a beneficial finding and an attempt on the part of the body to increase the strength of the bone at this, the greatest area of stress (fig 2). Farther down the shaft, on the lateral cortical wall where the stem is pushed laterally to prevent the prosthesis from tilting into coxavara, the cortical wall likewise thickens and the periosteum lays down 1 or 2 mm. of

increased bone (fig. 2). From the side wall, at the level of the lower end of the tip of the prosthesis, there begins to form a hooklike projection which curls about the lower tip of the prosthetic stem to anchor it firmly against the lateral wall (fig. 1). This usually is visible in the roentgenograms as early as three months following operation. This hook when formed increases in density and finally firmly immobilizes the prosthetic stem against the lateral cortical wall. In no instance has there been a tendency for the tip to cut through the lateral cortical wall because of pressure. Some tips have been driven through this side wall at the time of insertion by the operator hammering home the prosthesis instead of fitting it gently into the medullary cavity.

In not all prosthetic reconstructions does bone grow tightly around the stem. A radiolucent line of junction between dense bone and prosthesis is occasionally seen forming a bony cradle about the stem, indicating that some toggle motion still exists (fig. 2). This usually disappears in later roentgenograms as the reparative bone grows more snugly about the metal stem (fig. 1). One femoral shaft failed to calcify to a satisfactory density by the end of the ninth month. The exact reason for this is not clearly determined to date, since no evidence of infection nor of metastatic malignancy is apparent. Any splotchy decalcification or pitting should signify a low grade infection even in the absence of pain or fever. When the prosthetic stem becomes well anchored to the lateral cortical wall, there occurs a thickening and condensation of the medial wall at the same level. This was apparent at an earlier date with the large round stem model that was used as an early prosthesis. It is less noticeable in the present long, thin-stemmed model.

SUMMARY

The technique of operation developed during two years' experience with a Vitallium intramedullary hip prosthesis is described, together with remarks on technical difficulties to be anticipated and complications to be avoided.

The indications for the use of the prosthesis are given, and its prime indication as a method of salvage is stressed. Caution is urged against the indiscriminate use of this adjunct.

The results obtained in 25 cases are discussed. Nineteen have shown satisfactory results. Four failures occurred. Two were equivocal as to result. Complications leading to unsatisfactory results in all probability can be reduced drastically in the future as the result of this experience.

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ABSTRACT OF DISCUSSION

DR. EDWARD T. SMITH, Houston: Dr. Thompson has brought us a timely paper. For the past few years the use of a hip prosthesis has become popular. In fact, it has become so popular that there are some forty different types of prostheses described and used. In doing any type of reconstructive work, we orthopedists must keep in mind that we are trying to salvage an already pathologic condition. Dr. Thompson has listed in his paper a number of complications, and all of us who have had any experience with the use of a hip prosthesis have likewise experienced some bad results, or should I say, we did not get the good results we had hoped for. My reason for wanting to emphasize this phase of Dr. Thompson's paper is that there is a tendency for surgeons to become overenthusiastic with any new procedure and consider it a cure-all and a remedy for all their problems. I am glad to hear Dr. Thompson say that an arthrodesis of the hip is still a good procedure under certain conditions.

Concerning his criteria for a satisfactory prosthesis, I heartily agree with his ten points. A careful analysis of these ten points will help in determining the indications for the use of a hip prosthesis.

Dr. Thompson mentioned that one of the criteria for a satisfactory result is "how soon the patient discards his crutches and cane." I personally encourage my patients to keep their crutches and cane, particularly those in the older age group. I, for one, am for starting a campaign to encourage elderly people and people who have an unsteady gait to use a cane. It is just as necessary for these people to use an appliance to assist their walking as it is to use glasses to assist their sight or a hearing aid to assist their hearing.

There are many little tricks in Dr. Thompson's description of the operative technique which may be overlooked if one is not frequently performing surgery about the hip joint even though he may be familiar with the anatomy of the hip region. Remembering these points can mean the difference between an easy operation and a difficult operation and can also affect the operative results.

"MARCH OF MEDICINE" TELEVISION SERIES

"March of Medicine," a series of television broadcasts designed to create better understanding by the public of some of the major health problems and greater public awareness of the extensive research being carried out by the medical profession, has been resumed this fall. It is sponsored by the American Medical Association and Smith, Kline and French Laboratories.

The first of the series, presented October 8, featured discussion and demonstration of recent advances in coronary artery disease and hypertension. Those on the program included Drs. Herrman L. Blumgart, Howard B. Sprague, Paul D. White, and Robert W. Wilkins, all of Boston.

The November 5 program will be a progress report on cancer research and will be aired at 9 p. m., CST, over NBC-TV. The December 3 telecast will present highlights of the AMA annual clinical session in St. Louis.

The series is aired over approximately seventy NBC-TV network stations in the United States and also in Ottawa, Toronto, and Montreal.

CHRONIC OR PERIODIC HEADACHE

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HEADACHE, like all pain, is a warning that something is wrong within the person or in his relationship to the environment. Its common occurrence may lead many people to minimize the seriousness of a chronic headache. Neither should physicians underestimate its myriad economic and social implications in the life of the patient and the family. Disruption of the atmosphere of the home during the attack and the effect of chronic invalidism on the personality development of the growing child are combined with loss of earning power and untold medical expense. The latter can become exorbitant if the patient gets into the medical whirlpool of unnecessary referral to numerous specialists or else "shops about" with the multitude of nonmedical healers. Although the seriousness of chronic headache should not be underestimated, the symptom all too often assumes undue importance in the life of both the patient and his physician.

Headache is a challenging problem to all physicians, especially to the general practitioner. The overwhelming preponderance of the functional and psychogenic type of headache calls for an initial careful historical and routine physical evaluation. Recognition of this variety with institution of proper early treatment will prevent fixation of the headache and prevent waste. When the etiology cannot be identified in the earlier surveys, the practitioner is ideally situated to make appropriate referral, then to coordinate the treatment program.

Old theories for the cause of headache have been replaced by a more exact knowledge of the mechanism of head pain arrived at through scientifically controlled studies such as those of Wolff[3] and his coworkers. The task of making a specific diagnosis is now easier. Special methods of treatment based on this new knowledge have been devised. It was determined that most of the structures inside the cranium are insensitive to pain with the exception of the venous sinuses; the dural and cerebral arteries; a portion of the dura adjacent to the large arteries; the fifth, ninth, and tenth cranial nerves; and the upper three cervical nerves. Painful responses were elicited when these structures were stimulated, either by traction, displacement, inflammation, dilatation and distention, or else direct pressure on the previously mentioned sensory nerves.

In contrast, most of the extracranial structures, especially the arteries, were found to be pain sensitive. Excluding the eyes, ears, and sinuses, the basic mechanisms for extracranial head (not face) pain are, first, dilatation of the branches of the external carotid artery, and second, prolonged painful spasm of the muscles of the scalp, pharynx, and neck. If the original stimulus is not interrupted within the first few hours, secondary tissue edema results to prolong the pain. Any periodic or chronic headache, whether of intracranial or extracranial origin, has more than one mechanism involved in pain production.

This information has brought some orderly concept into the chaotic array of pains and other sensations commonly called headache. Also, it revealed that particular etiologic factors do induce a certain headache syndrome. This discussion is confined to the chronic or periodic headache and excludes the acute headaches with rather obvious cause.

Etiologically, headaches are of (1) the organic or (2) the functional variety. The former is associated with definitive structural changes either within or without the cranial cavity. A functional headache either is associated with temporary physiologic alterations or is entirely symbolic (psychic) in nature. Authorities variously estimate that organic factors produce between 15 and 20 per cent and functional and psychogenic causes account for the remaining 80 per cent of all long-standing headaches.

Again, the physician's approach to determining the cause of headache is enhanced by such a practicable diagnostic classification as that of Morrison[2] modified from the ideas of Wolff[3] and Moench.[1] The headache categories are based on etiology and listed in order of frequency of occurrence. To reverse Morrison's grouping and proceed from the least to the most frequent category, one finds histamine cephalalgia listed last, or eighth. Seventh in frequency is headache associated with intracranial disease; sixth, with ocular disease; fifth, with diseased nasal and paranasal sinuses; fourth, with systemic disease; and third, with diseased structures of the neck such as arthritis of the cervical spine, fibromyositis, and myalgia. All of these etiologic factors are of organic nature. The two remaining categories, vascular and psychogenic headache, encompass the headaches of a functional etiology.

From the Department of Neuropsychiatry, University of Texas Medical Branch.

Read before the Section on Eye, Ear, Nose, and Throat, Texas Medical Association, Annual Session, Houston, April 28, 1953.

ORGANIC HEADACHE

The physician must consider the organic probabilities in the study of each patient with chronic headache. The descriptive history of the headache may delineate a particular type. Questions as to the character of the pain, its location, its time of occurrence and duration, and factors which either aggravate or produce relief are helpful. The usual medical history including exposure to drugs, noxious agents, or trauma, must be examined in every case. A brief evaluation of the personality makeup with emphasis on the presence of excessive anxiety or resentment, prolonged stress, frustrations, and insecurities can reveal primary or contributing etiologic factors. A thorough general physical examination including the routine laboratory procedures is a necessary and invaluable aid in this diagnostic survey.

If migraine is suspected, simple diagnostic tests such as nitroglycerin, gr. 1/50 placed under the tongue will produce a typical hemicrania, which is then relieved by 1 cc. of dihydroergotamine injected intramuscularly. An injection of 1 per cent procaine into the tender scar area of the scalp in post-traumatic headache or into the tender point at the top of the trapezius muscle in myalgia will relieve pain and further help to establish that specific etiology. Of course, the most dramatic test of all is the injection of 0.5 to 1 cc. of 2.75 mg. ampule of histamine acid phosphate to produce a sharp, boring temporal and retro-orbital head pain, tearing and redness of the eye nearest the injection, and secretion from the corresponding nostril that are so classical of Horton's histamine cephalalgia. Histamine sensitivity is the rarest cause of headaches and occurs only in the presence of arterial disease, yet most headache veterans of the medical circuit, young or old, have been histamine tested and even desensitized.

The limits of this presentation preclude further emphasis on these significant diagnostic studies. Likewise, only brief mention can be made of the various causes and mechanisms of organic headache. The writings of Wolff, Moench, Von Storch, and Friedman offer an ideal reference source.

Although intracranial pathologic conditions cause only about 5 per cent of chronic headaches (Moench), the possible existence of brain tumor, vascular disease, inflammations, and sequelae after head injury should be ever before the physician. Such headaches are periodic until late in their course; may be relieved by mild analgesics; and usually are associated with signs of cerebral dysfunction in the form of convulsions or changes in the motor, sensory, speech, or visual sphere. When the physician is suspicious of intracranial lesions, it is desirable to seek proper consultation to determine the appropriateness of skull roentgenograms, electroencephalography, or the more specific air studies and/or angiography. These expensive tests are not to be considered as routine diagnostic procedures and should be done only when specifically indicated.

As with histamine sensitivity, ocular disease is a rare cause of headache, but most patients get the benefit of refraction all too early in the case study. Localized ocular diseases such as glaucoma and iritis produce orbital and retro-orbital pain. So do such refractory errors as hyperopia, astigmatism, and extra-ocular muscle imbalance (not myopia). Headache may occur after use of the eyes. Also, secondary myalgia in the neck muscles often contributes to ocular headache.

The pain of ear disease requires no special mention except that, as in disease of the nasal and paranasal structures, a history of a preexisting infection of the upper respiratory tract is elicited. Thankfully, the era is passed when multiple nose and sinus surgery flourished as the cure for persistent headaches. MacAuliffe, Goodell, and Wolff studied the pain sensitivity of nasal and sinus structures. They concluded that inflammation with engorgement of the turbinates, the superior nasal spaces, and the sinus ostia caused most of the pain. Headache unassociated with these changes is not due to nasal or sinus disease. Also, if facial pain, frontal, temporal, or vertex headache is not helped by application to the turbinates and sinus ostia of a vasoconstrictor and weak anesthetic solution, it is not due to nasal disease. Other symptoms of sinus disease confirm the diagnosis. Again, secondary reflex spasm of the muscles of the scalp and neck contribute to the severity of nasally induced head pain.

The systemic factors in chronic headache require careful delineation. Chronic fever, hypoxia of whatever origin, and functional hypoglycemia produce dilatation and distention of the branches of the internal carotid artery similar to the effects of histamine. Like migraine, the headache associated with arterial hypertension is caused by dilatation and distention of the branches of the external carotid and dural arteries. Wolff has shown that the term "hypertensive headache" is misleading because the headache is not directly related to the level of the blood pressure.

FUNCTIONAL HEADACHE

Headaches of functional etiology comprise the remaining categories in Morrison's classification. Headaches of this second large subdivision are of the (1) purely vascular and (2) psychogenic variety. Examples of the vasodilator and distention type of headache are migraine, histamine cephalalgia, fever, alcoholic intoxication, and the postepileptic seizure state.

The psychogenic mechanisms for headache are (1) anxiety-induced autonomic dysfunction with vascular dilatation and reflex myalgia; (2) the simple depression with myalgia; and (3) the symbolic type found in hysteria and the psychoses.

Migraine, the most important of the vascular headaches, has been discussed so thoroughly in recent literature that only a few practical points will be stressed. Moench's four classic signs of true migraine should exist before making such a diagnosis:

1. Severe hemicrania.
2. Gastrointestinal upset; usually nausea and vomiting.
3. Visual aura preceding or occurring in conjunction with the headaches.
4. History of a familial tendency and often of an allergic background.

With the family history and the kinship of migraine to epilepsy, some patients have abnormal electroencephalographic tracings. Anticonvulsants like Dilantin Sodium or Mesantoin, in the usual dosage, are sometimes useful in prophylaxis. Because psychogenic forces are rampant within the personality structure of the migraine patient, further prophylaxis is directed along psychotherapeutic lines. Estrogen therapy was of little value in Von Storch's series, but attention should be given to the patient with frank allergy.

Symptomatic treatment of migraine is based on efforts to reduce the amplitude of pulsation in the dilated and distended external carotid and dural arteries. If administered early, ergotamine tartrate, dihydroergotamine by hypodermic or such oral preparations as Cafergot (which with caffeine potentiates the effectiveness of ergot as a vasoconstrictor), Valoctin (a sympatheticomimetic vasoconstrictor), or Fiornal (an analgesic, vascoconstrictor, and sedative) may abort an attack. Should the headache progress for several hours, edema develops in the vessels and supporting tissues and produces secondary spasm of scalp and neck muscle to prolong the headache.

Hence, it is found that myalgia is a concomitant of ocular and nasal disorders, neck lesions, post-traumatic headaches, functional vascular headaches, and those psychogenic headaches that induce physiologic vasodilatation and reflex muscle spasm. The pain is most intense in the region of the neck, shoulders, and occiput. It may spread over the entire head. The patient describes stiffness, pain, and soreness in the neck; tenderness over the upper border of the trapezius muscle; and a numbness of, or even a highly sensitive, scalp with a band or caplike sensation about the head. Wolff, by use of the electromyogram, has been able to demonstrate a definitive spasm of the muscles in the region. A careful history directed toward an analysis of the known factors in headache production help in the differential search for the prime etiology.

Actually, this is a description of the popular "tension headache" of psychogenic origin. Such headaches are an early and habitual symptom of the simple depressive reaction and the anxiety psychoneurosis. Although its cause is psychogenic, the pain or discomfort results from alterations in the physiology of the muscles of the face, scalp, neck, and shoulders, plus minimal dilatation of the extracranial arteries.

Functional vascular headache and organic head pain are consistently periodic. In contrast, the *psychogenic* headache usually is constant throughout the waking hours, exists for months or years, and is not influenced by forces altering the intracranial hydrodynamics. True vasodilator headaches often are intensified by a drink of alcohol, heat, or hunger. Such response is rare in psychogenic headaches except for the migrainous-like headache sometimes found in the anxiety state which, through autonomic dysfunction, is associated with cerebral vasodilatation. Here, headache, although perhaps the chief complaint, is but one of many symptoms of illness. Numerous symptoms of personality dysfunction are uncovered. The diagnosis of an anxiety psychoneurosis or a mild depression, or for that matter any other psychogenic reaction, should be made on the presence of well established, positive diagnostic criteria and not by just the process of excluding organic disease.

Therapeutically, it is important to differentiate the anxiety state from a depression. Patients with an anxiety state experience great anxiousness, may be phobic, exhibit restlessness and muscular tension, and show periods of autonomic nervous system dysfunction including tachycardia, gastrointestinal disturbances, and excessive sweating. The degree of reaction is tuned acutely to external stress. The personality survey reveals the dynamic environmental forces that produced the illness. Many patients readily are receptive to a logical explanation of the mechanism of the headache and the meaning of other symptoms. They are greatly benefited by discussion which gives reassurance and allows them to ventilate or correct their problems. Simple sedation, preferably a combination analgesic, vasoconstrictor, and sedative, gives palliative relief of the tension headache during the treatment program. Barbiturates should be prescribed under strict control. Extreme caution must be exercised in administering narcotic injections to a chronic headache patient.

Too many depressed patients with a tension headache go undiagnosed for months or years. Otherwise, a miserable existence, unnecessary expense, loss of earning power, and even suicide could be prevented. The symptoms of the mild depression, unlike the anxiety state, are not readily influenced by the environment. All complaints, especially headaches, are

worse in the morning. Because of the characteristic insomnia of this illness, the patient believes that the headache awakens him. Likewise, the patient attributes to the headache his loss of zest and energy and of interest in sex, food, and the usual daily pursuits. Brooding, worry over trifles, despondency, and feelings of futility are present in variable degree. The etiology of a depression is endogenous and treatment limited to psychotherapy is impracticable in many cases. The management of each case must be decided upon individual features. Supportive psychotherapy, attention to the physical health needs, mild daytime sedation, and the precaution to issue a limited quantity of sleep-producing drugs are recommended. The suicide potential is a constant threat. Administration of electroshock to the depressed patient with a long-standing tension headache can and does produce gratifying results.

Another mechanism for a psychogenic headache is to be found in symbolism. This is purely psychic, is not associated with disordered physiology, and represents the conversion or displacement of unconscious conflicts or repressed hostility into the word symbol "headache." The presence of a headache sensation and the protection it affords as a mode of retreat from the demands of society is a form of defense frequently encountered as a forerunner of a psychosis. The intensity of the patient's anxiety, the complaint of exquisite head pain, and the patient's persistent demands for relief, usually a hypodermic injection, should cause the physician to suspect an incipient psychosis. The best solution is referral for specific, although too often unfruitful, psychiatric treatment.

The readily recognizable bizarre description of the headache of a patient with conversion hysteria is supplemented by the obvious gain of the complaint. The hysterical patient shows signs of being in severe distress and great suffering, but underlying all of this is an air of indifference. The symptom serves to solve emotional conflicts and to manipulate and control the life situations. Drugs are inadvisable, although suggestive measures and the physician's assistance in solving the immediate problem may resolve the symptom. For the general practitioner treatment is symptomatic; for the psychiatrist, it calls for management therapy and, in a few selected instances, uncovering or insight psychotherapy.

SUMMARY

Less than 10 to 20 per cent of all patients with chronic headaches have an organic basis for their symptom. Functional headaches constitute 80 per cent of headaches and are of the vascular and psychogenic variety.

An attempt is made to describe, delineate, and suggest therapy in each of the headache categories.

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ABSTRACT OF DISCUSSION

DR. STEPHEN WEISZ: Dallas. The authors deserve commendation for their lucid presentation of such a complex subject. As they have pointed out, organic headaches constitute less than 20 per cent of the chronic and periodic headaches. The overwhelming majority of these headaches belong to the functional group. The differential diagnosis between organic and functional types of headache generally is much less difficult than the differentiation of the two main forms of functional headache, namely, the vascular and tension types. It is well to keep in mind that these two varieties often mix and overlap. For instance, in hypertensive persons we find both tension and vascular headaches, although the tension type is usually the dominant variety. As another example, a decrease of the typical vascular headaches frequently takes place in migraine patients as they grow older and is replaced by a different kind which has the earmarks of the tension type. The same is true for most patients who tell us that they have "sinus." A more complete study of their complaints many times reveals that in reality they refer to head pain rather than to a pain based on sinus disease.

I should like to underline that in all the headaches mentioned there is a physiologic disturbance of the peripheral end organs, whereas, in contrast, the headache occurring in hysteria represents a symbolic expression of a mental conflict. Only the correct evaluation of the type of headache present will facilitate the proper management. In this respect, I should like to reiterate the authors' statement that the treatment of headaches is difficult and at times disappointing. It is one thing to take care of the headache when it is present and another to avoid its recurrence. It is in the latter category that psychiatric assistance can be of benefit. A careful psychiatric survey will give information that will allow the evaluation of all factors involved and may help to reduce those factors responsible for the precipitation of the recurring attacks. I found electroshock the most beneficial form of treatment in the so-called intractible headache. In this condition, which appears to be a fixed tension type, various trigger mechanisms induce severe headaches during which the patient has extraordinary discomfort and visibly suffers a considerable intensity of pain. It usually does not yield to any form of medication unless one approaches measures resembling narcosis. The frequency with which physicians, regardless of their specialty, encounter headaches makes this presentation welcome.

Blood Collection Centers Closed

The American Red Cross is closing fifteen of its blood collection centers as a result of the truce in Korea; it also is terminating contracts with seventeen community blood banks. Announcement of this cutback was made by the Office of Defense Mobilization. The Red Cross will continue to collect blood at its forty-five regional centers to meet military demands, to supply civilian users, and to stockpile derivatives for use in disasters.

TREATMENT OF URINARY INFECTIONS

Choice of Drug

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IN the past few years we have witnessed a vast change in the treatment of urinary infection because of the discovery of new and potent antibiotics and chemotherapeutic agents. Broadly speaking, they have revolutionized the course of urinary infections. To obtain these good results, however, an understanding of their application and limitations is necessary.

This discussion will be confined to (1) the demonstration that the drugs now available have a definite selectivity for inhibition of certain organisms, (2) an indication of which drug is most effective for the organism causing the infection, and (3) a brief discussion of the treatment for certain clinical conditions.

SELECTIVITY OF DRUGS

There are ten organisms which are commonly found to cause infection in the urinary tract (fig. 1). These are Escherichia coli, Escherichia intermedium, Aerobacter aerogenes, Paracolobactrum coliforme, Pseudomonas aeruginosa, Proteus vulgaris, Alcaligenes fecalis, Streptococcus fecalis, Staphylococcus, and Streptococcus.

There are a number of drugs used to inhibit these organisms, the most popular of which are penicillin, streptomycin, Aureomycin, terramycin, Chloromycetin, polymixin B, Mandelamine, and Gantrisin. Two drugs more recently introduced are an antibiotic, Erythromycin, and a chemotherapeutic, Furadantin, which are indeed useful in selected cases.

The selectivity of these drugs for the aforementioned organisms is demonstrated in figure 2 and table 1, which present the in vitro results of a study made on 1,000 organisms isolated from actual urinary infections in the clinic with which I am associated.

Pseudomonas aeruginosa.—One of the most striking examples of this selectivity is the effect on the Pseudomonas. Terramycin and Aureomycin will inhibit about one-third of the strains. In contrast, Chloromycetin inhibits only 9 per cent and Gantrisin 6 per cent. Figure 2 indicates the detailed results of this in vitro study.

Proteus vulgaris.—Studies on the Proteus also show this selectivity. Chloromycetin inhibits 96 per cent of the strains and Gantrisin inhibits 70 per cent. On the other hand, only 7 per cent are controlled by Aureomycin and 6 per cent by terramycin (fig. 2).

Aerobacter aerogenes.—Aureomycin is effective in 88 per cent of the strains of Aerobacter, and terramycin in 77 per cent. In contrast, Gantrisin affects only 8 per cent and Chloromycetin 36 per cent (fig. 2).

Escherichia coli.—The colon bacillus is the most common organism found and fortunately is susceptible to all the chemotherapeutic agents and antibiotics. From these findings, however, it is apparent that certain drugs are more effective against some organisms and totally ineffective against other organisms (fig. 2).

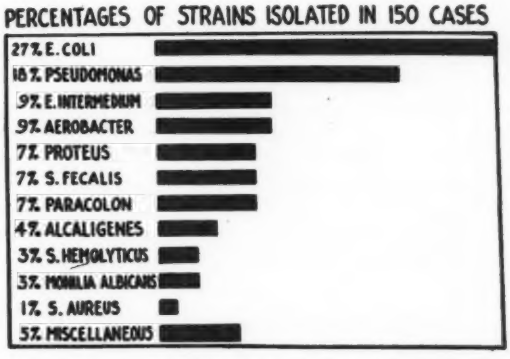

FIG. 1. Organisms found in urinary infections in the order of their frequency of occurrence.

EFFECTIVENESS OF DRUGS

Aureomycin, Terramycin, Chloromycetin, and Gantrisin.—Table 1 gives the detailed picture of the effectiveness of four popular drugs against organisms isolated from infections in the urinary tract. In addition to the organisms mentioned above, the chart demonstrates the following:

The Escherichia intermedium is highly susceptible to Aureomycin and to a lesser degree to Chloromycetin.

The Alcaligenes fecalis is susceptible to Aureomycin and terramycin, but only mildly so to Gantrisin and Chloromycetin.

The Paracolon is inhibited by Aureomycin in 88 per cent of the strains, but is inhibited by Chloromycetin and Gantrisin in only 50 per cent.

Read before the Section on General Practice, Texas Medical Association, Annual Session, Houston, April 28, 1953.
This study was made possible at St. John's Hospital by a grant from the Olin Foundation.
**Associate Clinical Professor, Department of Urology, St. Louis University.*

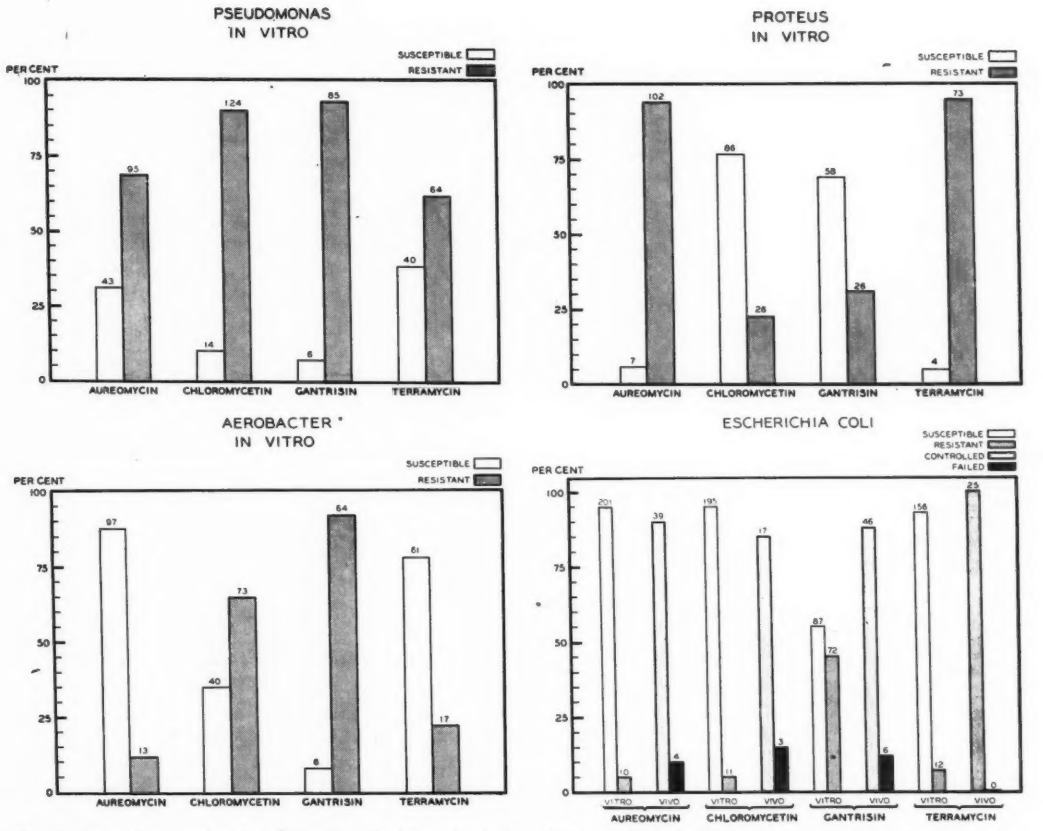

FIG. 2. Charts demonstrating the comparative effectiveness in vitro against Pseudomonas, Proteus, Aerobacter, and Escherichia coli infections of Aureomycin, Chloromycetin, Gantrisin, and terramycin. It will be noted that for Escherichia coli, the effectiveness of the drugs in patients parallels the laboratory sensitivity tests.

The Streptococcus fecalis is very susceptible to Aureomycin but is resistant to Gantrisin in 60 per cent of the cases and to terramycin in 25 per cent. This organism also is highly susceptible to Mandelamine.

The Staphylococcus infections deserve reappraisement. Heretofore penicillin has been the drug of choice in Staphylococcus infection, but the drug has been used so universally that it now has diminished in its effectiveness since most of these infections are caused by the surviving resistant strains. Many reports are coming from the larger clinics indicating resistant strains to be as high as 70 per cent of the total staphylococcic organisms. Of 14 consecutive strains of Staphylococcus and Streptococcus isolated in our clinic, 12 were resistant to penicillin.

This abuse of the use of penicillin and the prescribing of it for all infections is unwarranted. In one naval flotilla off the coast of Korea an official order required all sailors to take penicillin daily as a prophylactic measure, so that in many instances it has been prescribed for not only *all* infections, but even for *no*

TABLE 1.—*In Vitro Sensitivities to 4 Drugs of 1,000 Organisms Isolated in Urinary Infections.*

Organism	No.	Aureomycin		Terramycin		Chloromycetin		Gantrisin	
		Susceptible	Resistant	Susceptible	Resistant	Susceptible	Resistant	Susceptible	Resistant
Escherichia coli	245	201	10	156	12	195	11	87	72
Escherichia intermedium	59	51	2	24	21	39	9	12	34
Aerobacter aerogenes	122	97	13	61	17	40	73	6	64
Paracolobactrum coliforme	31	26	4	13	4	15	11	13	7
Pseudomonas aeruginosa	159	43	95	40	64	14	124	6	85
Proteus vulgaris	117	7	102	4	73	86	26	58	26
Alcaligenes fecalis	38	30	2	25	2	17	15	9	22
Staphylococcus	100	60	10	43	35	60	9	18	31
Streptococcus fecalis	81	77	0	44	21	56	10	10	42
Fungus infections	48	—	—	—	—	—	—	—	—
Total	1,000								

infection. Erythromycin seems to be taking us out of our dilemma, however.

Erythromycin.—Erythromycin is highly selective in its effectiveness, that is, it affects the coccal infections only and is totally ineffective against the bacillary infections. It is well tolerated, does not develop resistance as quickly as penicillin, is easily administered by mouth, and inhibits the strains of Staphylococcus and Streptococcus which are resistant to penicillin (table 2).

Furadantin.—Furadantin is a chemotherapeutic agent, a nitrofuran recently introduced for study and now, within the past few months, made available commercially to the profession. I have made a study of this drug and believe it to be very useful. It is administered by mouth and readily absorbed in the intestinal tract. The Proteus is highly susceptible to Furadantin. In 25 patients treated with Furadantin the Proteus infection was controlled; in 4 other patients treated the infection was not controlled. The

TABLE 2.—*Sensitivity of Organisms to Erythromycin.*

Organisms	No. Strains Tested	Inhibition Concentration (gamma/cc.) Range	Average
Micrococcus pyogenes var. albus* (Staph. albus)	13	0.4-2.0	1.1
Micrococcus pyogenes var. aureus (Staph. aureus)	1	0.8	0.8
Streptococcus pyogenes†	2	0.08-1.2	0.64
Enterococcus‡			
Streptococcus fecalis	8	0.6-4.0	1.42
Streptococcus durans	1	0.4	0.4
Proteus	9	>200	>200
Pseudomonas	16	>200	>200
Escherichia coli	20	>200	>200
Aerobacter aerogenes	4	>200	>200
Intermediate coliforms	7	>200	>200
Alcaligenes fecalis	2	>200	>200
Total	83		

*12 of the 13 strains were found to be resistant to penicillin by the disk plate method.

†1 strain was resistant to penicillin; 1 strain was slightly sensitive to penicillin.

‡1 strain was slightly sensitive to penicillin; the other 8 strains were resistant to penicillin.

Escherichia coli, the Aerobacter aerogenes, the Paracolobactrum, and the Alcaligenes also were controlled somewhat by the drug. The effect on the Pseudomonas has been disappointing; only 1 was controlled out of 21 treated.

When massive doses were given to rats, degeneration of the seminiferous tubules of the testes was noted, but complete recovery occurred when the drug was withdrawn. Intensive laboratory studies over a two year period by the manufacturer indicate that the degenerative process is one of metabolic arrest, rather than toxic effect. In our clinic we have administered the drug to some 80 patients with no serious toxic effects demonstrated. Nausea is a prominent feature of this drug, but it is somewhat controlled by giving the drug after meals and with an alkali. Four patients have received this drug for twenty days with no ill effects.

Polymixin B.—Polymixin B studied in vitro was found to inhibit a higher percentage of strains than any other drug now available. The nephrotoxic and neurotoxic effects are reported by E. J. Pulaski, who stated: "When a daily intramuscular dosage did not exceed 2.5 mgm per kilogram, there was no increase in the blood urea, nitrogen, or nonprotein nitrogen, nor was there any clear-cut evidence of nephrotoxicity in the form of tubular damage attributable to the drug. Neurotoxic symptoms disappeared in every instance within twenty-four hours after the last dose was administered."

We have limited our use of the drug to severe cases when the organism is not sensitive to other antibiotics, since local pain, dizziness, weakness, peripheral parasthesias, and pain along the nerve trunk may be expected in some patients. These symptoms disappear within twenty-four to forty-eight hours after discontinuance of the drug.

Choice of Drug.—The choice of drug depends on the judgment of the attending physician. The physician must weigh the following: (1) the ease of administration, (2) the toxic symptoms and side reactions, (3) the development of resistance, (4) the cost, and (5) the effectiveness.

Table 3 may be helpful in the choice of treatment.

TABLE 3.—*Drug of Choice in Treatment of Urinary Infections.*

Organism	1st Choice	2nd Choice
Escherichia coli	Gantrisin	Mandelamine
Escherichia intermedium	Terramycin	Aureomycin
Aerobacter aerogenes	Aureomycin	
Paracolobactrum coliforme	Aureomycin	
Pseudomonas aeruginosa	Terramycin	Streptomycin or Polymixin B
Proteus vulgaris	Furadantin	Chloromycetin or Gantrisin
Alcaligenes fecalis	Terramycin	
Streptococcus fecalis	Mandelamine	
Staphylococcus	Erythromycin	Penicillin
Streptococcus hemolyticus	Erythromycin	Penicillin

CLINICAL CONDITIONS

Thus far, drugs and bacteria have been discussed. A few remarks may be made from the clinical viewpoint about the various diseases encountered.

A patient seen for the first time with an acute urinary infection, showing pus in the urine, urgency and frequency of urination, and possibly chills and fever, may be treated with one of the conservative drugs, such as Gantrisin or Mandelamine; probably by Aureomycin if the symptoms are severe. An examination of voided urine in the male and a catheterized specimen in the female should be examined microscopically, a stained smear made, and a culture grown if possible. Sensitivity tests should be done when possible.

Acute Cystitis.—Acute cystitis in women is really a urethrocystitis, striking the patient suddenly with urgency and frequency of urination and even hematuria. Usually a colon bacillus is responsible for such infections, which are easily controlled by any of the urinary agents. The urethra should be dilated with sounds when the condition is better. Urethral suppositories composed of 1 per cent Protargol give much relief. Endoscopic application of 10 per cent silver nitrate often is effective in granular urethritis of women.

Pyelitis of Pregnancy.—When pus persists in the catheterized specimen of a pregnant patient, the physician should visualize the probability that one or both of the ureters are dilated and atonic, bringing about the retention and infection of urine in the renal pelvis. The treatment of choice is medication and placing the patient in the knee-chest position, but should this fail an indwelling catheter may be necessary to promote active drainage from the pelvis of the kidney. The physician should not wait too long to impose this catheter or else the kidney may be irretrievably damaged (fig. 3a).

Incrustations.—One of the most serious complications of indwelling catheters or nephrostomy tubes is the formation of incrustations within the catheters, causing them to block. The vast majority of these incrustations are caused by the urea-splitting organisms, the Proteus or the Staphylococcus. Any infection which is persistently alkaline despite the administration of ammonium chloride or acid ash diets may be assumed to be Proteus infections, and Gantrisin, Chloromycetin, or Furadantin should be administered.

Pyelitis in Children.—Any urinary infection found in infants or children is too often called pyelitis. All resistant or persistent infections in children deserve careful cystoscopic examination to discover the cause of obstruction which is always present. Bladder neck obstructions are quite common and will seriously impair renal function unless corrected (fig. 3b and c).

CONCLUSION

In the treatment of all resistant or persistent urinary infections two diagnostic objectives are indicated: (1) the identification of the infecting organism and (2) the location and type of obstruction causing the persistence of infection. Upon this depends the treatment, which consists of the relief of the obstruction and the choice of drug according to the type of organism isolated.

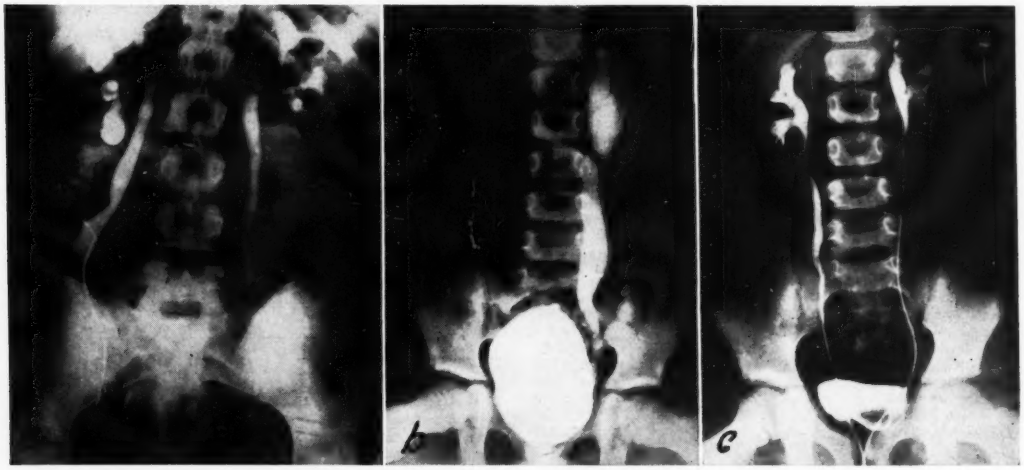

FIG. 3. Roentgenograms of patients with urinary disease.
a. Pyelitis of pregnancy with atonic dilated ureters and renal pelvis. Catheter splinting sometimes is necessary.
b. A cystogram producing a ureterogram with dilation and decreased function due to bladder neck obstruction in a child.
c. The patient in *b* after restoration to normal of the ureters and pelvis following resection of the bladder neck.

ABSTRACT OF DISCUSSION

DR. ABEL J. LEADER, Houston: Dr. Carroll's presentation is an unbiased report based on an enormous effort in the laboratory. We can therefore with profit to ourselves and our patients do well to remember the lesson he teaches: that there is a definite selectivity in the action of the various antibiotics and chemotherapeutic agents used in the treatment of urinary infections. No one "wonder drug" works equally well in the eradication of infection produced by the nine organisms cited. It has become increasingly more important for us to identify the causative agent in these infections in order that the infection be managed most effectively. We would be wise, I think, to regard the appellation "broad spectrum antibiotic" as the brain child of some enterprising advertising executive.

These "wonder drugs" have provided us with a fine opportunity for sweeping a lot more dirt under the proverbial carpet. Where infection keeps recurring after apparently satisfactory responses to one or more of these agents, we

must suspect the existence of an obstructive factor that serves to perpetuate the infection. This will require an investigation of the entire urinary tract. Such an investigation will entail cystoscopy with catheterization of the ureters to obtain fractional urine samples from each kidney for routine analysis, pyogenic culture, and possibly guinea pig inoculation. Renal function studies also will be carried out and retrograde pyelograms made. The omission of such an investigation may constitute a great disservice to the patient because it may prevent the discovery of a lesion at a time when its detection might lead to reparative surgery.

It is also important to remember that subsidence of symptoms does not necessarily indicate that the infection has been eradicated. Recrudescence of an acute infection is possible without a corresponding return of the original symptoms. If the infection is not discovered and adequately treated, the initiative may be lost and the infection may go on to stubborn chronicity. As criterion for cure of an acute urinary tract infection, the minimum requirement should be at least two negative urinalyses with absence of bacteria on gram-stain smears of the urinary sediment. These should be done one and two weeks after the subsidence of all symptoms.

There are occasions on which the clinician finds that he is dealing with an infection caused by an organism resistant to all of the available antibiotic agents and sulfonamide drugs. For such infections the value of the older therapeutic agents such as methenamine (Urotropin) and mandelic acid should not be forgotten. These may be effective when all else fails. Both require acidification of the urine to a hydrogen ion concentration of 5 or less, and this may be accomplished by giving 5 to 8 Gm. of ammonium chloride daily. It should be remembered that when used, these medications will be most effective if the fluid intake is sharply curtailed. Although such dehydration seems to violate a basic urologic principle, it is a necessary restriction. The daily dose of methenamine is 4 to 5 Gm. daily, given as divided doses. Mandelic acid is best given as the syrup of ammonium mandelate in 15 cc. doses at five to six hour intervals, each cubic centimeter containing 3 Gm. of mandelic acid.

LOWER NEPHRON NEPHROSIS

Emphasis on Conservative Treatment

SHERMAN B. LINDSEY,* M.D.; RICHARD D. HAINES,† M.D.; and VINCENT J. SIMMON,† M.D., Temple, Texas

LOWER nephron nephrosis is an entity characterized by the sudden onset of oliguria or anuria followed by acute renal failure and is usually associated with tissue or blood destruction. The precipitating conditions are frequently accompanied by shock. Lower nephron damage and the presence of heme casts in the lower nephron and collecting tubules are the main pathologic changes (fig. 1). Extensive studies have been made of lower nephron nephrosis cases during the past ten years.[2, 8, 9] The number of precipitating conditions in which lower nephron nephrosis has been reported is increasing (table 1).

The exact cause of renal failure in patients with lower nephron nephrosis is unknown. It is probably due to a combination of factors. Possible mechanisms of renal failure include renal ischemia, nephrotoxic action of certain substances released during tissue and blood destruction, obstruction of renal tubules by heme compounds, and allergic factors. The hypothesis that renal ischemia is the primary cause of tubular damage and renal failure in lower nephron nephrosis is the most generally accepted explanation at present.[2, 9, 13] Although impaired selective reabsorption seems to be the best explanation for oliguria or anuria present in lower nephron nephrosis, tubular obstruction by heme casts and decreased glomerular filtration may play a part.[8]

The general clinical course after the initial tissue insult follows a rather definite pattern. Shock is an early sign in most instances. After recovery from shock, the patient may not appear to be in great danger. However, signs of renal failure begin to develop during the first twenty-four hours and may progress to anuria regardless of increased intake of fluids. Frequently, the urine appears dark on the first and second days because of the presence of heme compounds. The urine has a low specific gravity and is acid. Albuminuria persists in various amounts during the illness. Vomiting may be an early sign. Uremia develops after the first few days and progresses rapidly. This is accompanied by a rise in the potassium, creatinine, and uric acid blood levels. The carbon dioxide combining power is usually decreased. The blood pressure begins to rise on the second and third days. Peripheral edema may develop during the illness. Pulmonary edema has frequently been reported as a terminal event. Cardiac arrest from hyperpotassemia has been suspected as the immediate cause of death in some patients.[15] Those that recover develop a marked diuresis followed by clinical improvement and decline in blood urea.

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†Department of Internal Medicine of the Scott and White Clinic.

PROGNOSIS

Death due to lower nephron nephrosis in crush injuries usually occurs by the seventh day.[8] Most patients with fatal transfusion reactions die by the tenth day.[8] Those who recover develop a sudden diuresis and gradual return to normal of results in renal function tests. It has been shown that results of renal function tests may be abnormal for several months.[2] Lucké[8] estimated the mortality rate of patients with lower nephron nephrosis developing acute renal failure and anuria as 90 per cent. Recent changes in methods of treatment may reduce this rate. Muirhead[11] reported the death of 4 persons in a series of 12 patients having extreme renal failure following incompatible blood transfusions. These patients were treated conservatively.

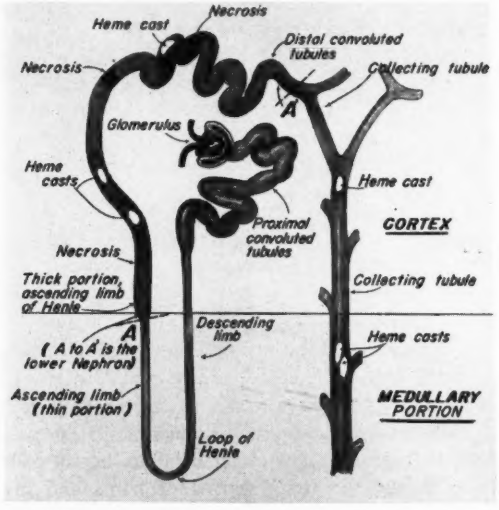

FIG. 1. Diagram of the nephron showing locations of necrosis and heme casts in lower nephron nephrosis.

PATHOLOGIC CHANGES

Gross Changes in Kidneys.—The kidney appears soft, pale, and swollen. The weight is usually increased. On examination of the cut surface, the cortex appears pale and the medulla is dusky.

Microscopic Changes in Kidneys (fig. 2): The first change noticed is a fine fat vacuolization in the cells of the ascending limb of Henle's loop.[9] By the third day, focal degeneration and necrosis of the thick limb of Henle's loop and distal convoluted tubules appear. Heme casts are found in the structures just mentioned and also in the collecting tubules. The stroma shows edema and the presence of inflammatory cells, usually lymphocytes and histocytes. Venous thrombosis also may be present near damaged tubules.

Changes in the upper nephron are slight. Slight dilatation of the proximal tubules and Bowman's capsule may be seen. The epithelial lining of Bowman's capsule may tend to the cuboidal. Hypertrophy of the juxtaglomerular apparatus and increased granularity of its cells have been reported.[8]

Regeneration of the tubular epithelium has been observed on the fourth day, and it is almost complete by about the tenth day.[8]

TREATMENT

Treatment may be divided into three phases: (1) immediate treatment phase, (2) treatment in the oliguric and anuric phase, and (3) treatment in the postanuric phase. The aim of treatment in all stages is that of maintaining fluid and electrolyte balance.

Immediate Phase

Proper treatment immediately may prevent fatal complications and renal damage. Shock should be treated promptly. Properly matched whole blood is the intravenous fluid of choice when hemorrhage or severe intravascular hemolysis occurs. Washed red cells may be used when limitation of fluid intake is indicated. Muirhead[11] has suggested toluidine blue or ACTH for the bleeding tendency associated with transfusion reactions. Local tissue damage should receive early surgical attention.

Alkalinization of the urine immediately following transfusion reactions and traumatic injuries has been recommended in the past. Maintenance of electrolyte

TABLE 1.—*Conditions in Which Lower Nephron Nephrosis Has Been Reported.*

Trauma
Crushing injuries
Battle wounds (mine explosions, gunshot wounds, blast injury, and severance of large blood vessels)
Major abdominal surgery
Burns (chemical and thermal)
Twisted ovarian cyst
Rupture of the uterus
Trauma of labor in pregnancy
Retroplacental hemorrhage
Electrical injury to muscles
Nontraumatic conditions
Blood transfusion reactions
Favism
Sulfonamide reactions
Heat stroke
Hyperpyrexia following intravenous typhoid vaccine therapy
Blackwater fever (falciparum malaria)
Hemolytic anemia of undetermined etiology
Eclampsia
Infections: acute pancreatitis, rickettsial diseases, severe streptococcal infections, and infantile gastroenteritis
Mussel and mushroom poisoning
High altitude anoxia
Anaphylactic shock
Paralytic myohemoglobinuria in man
Some cases of interstitial nephritis and hepatorenal syndrome similar to lower nephron nephrosis
Intravascular hemolysis following irrigation of bladder during transurethral resections
Chemical poisons: arsenicals, carbon tetrachloride, alkali, carbon monoxide, adulterated alcohol, isopropyl alcohol, phenol, photodeveloper, quinine, and phenyl hydrazine
Pulmonary infarction

balance is emphasized by most workers instead of alkalinization of the urine.[3, 12] Attempts to alkalinize the urine in acute renal failure may lead to pulmonary edema and alkalosis.[3]

Oliguric and Anuric Phase

Treatment in the second phase is based on the assumption that renal damage is reversible. If the patient can be kept alive for one or two weeks, enough tubular regeneration may be present for survival.

Fluid Balance.—Fluids should be limited to the replacement of daily fluid losses. Most investigators suggest a daily intake of 1 liter to replace that lost from the skin and lungs. In addition to this intake, fluid lost in the urine is replaced quantitatively. Additional fluid administration will be required if there is vomiting, diarrhea, fever, profuse sweating, or other abnormal fluid losses. Daily intake and output of fluids should be carefully observed.

Administration of enough fluid to induce edema in this phase in an attempt to dilute toxins has been tried by some investigators.[7] We believe there would be danger of precipitating pulmonary edema if this method were used.

Parenteral Therapy and Electrolyte Balance.—When oral or gastric tube feedings are not practical, fluid can be given intravenously as glucose in distilled water. Strauss[15] recommended 15 per cent glucose in distilled water in this condition. Hypertonic solutions have the advantage of supplying an adequate caloric intake on a limited fluid program. However, such solutions should be given slowly to prevent acute pulmonary edema. Electrolytes are added to the glucose as needed.

Acidosis can be treated with one-sixth molar lactate solution or 5 per cent sodium bicarbonate solution. Hypochloremia can be treated with isotonic sodium chloride solution. If the sodium level is excessive, 0.8 per cent ammonium chloride may be substituted for sodium chloride. If potassium poisoning develops, the potassium intake should be eliminated. Insulin and glucose have been suggested when hyperpotassemia occurs.[4] The use of cation exchange resins in hyperpotassemia associated with acute renal failure needs further study before being generally adopted.[10] Occasionally, calcium deficiency will appear in acute renal failure and can be treated with calcium gluconate.

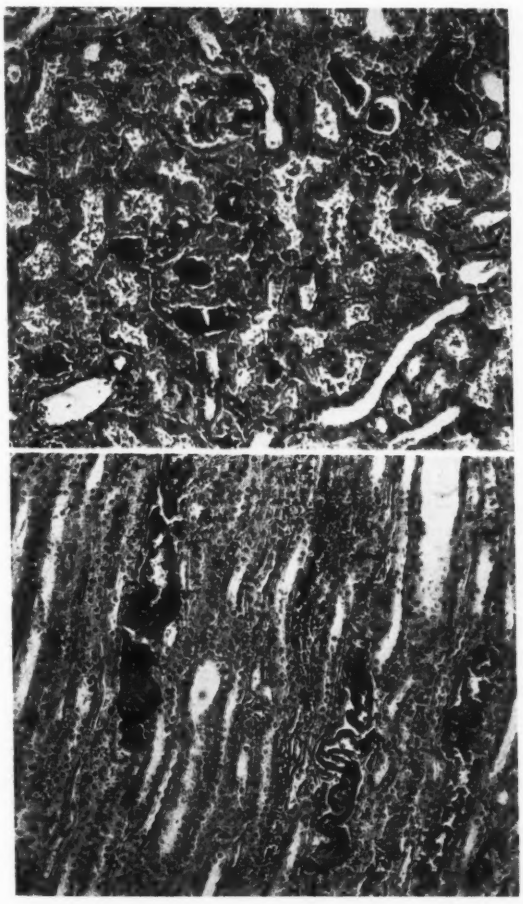

FIG. 2. Photomicrographs showing lower nephron nephrosis from transfusion reaction.

Above. Cortical region. Note the heme casts and changes in the epithelium of the distal convoluted tubules. x 135.

Below. Medullary region. Note the heme casts in the collecting tubules. x 135.

Photomicrographs were made by Dr. D. S. Morris, formerly of the Department of Surgical Pathology, Scott and White Clinic.

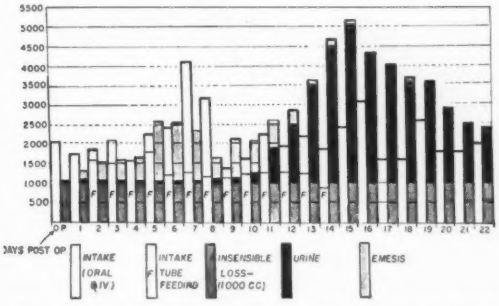

FIG. 3. Case 1. Graph showing daily intake and output of fluid in lower nephron nephrosis.

Vitamins should be added to the intravenous fluids or oral feedings. Plasma or serum albumin may be used cautiously if hypoproteinemia occurs. Severe anemia should be treated with properly matched blood or washed red cells. Digitalis may be necessary if congestive heart failure develops.

Frequent determinations of blood electrolytes are necessary to maintain electrolyte balance. Plasma pro-

tein levels and blood counts should be obtained every few days. Electrocardiograms may be helpful in detecting a deficiency or excess of potassium.

Oral Feedings.—If the patient is able to eat or will tolerate a gastric tube, a low protein, high carbohydrate, high fat diet is recommended to keep the production of nitrogenous products at a minimum. This also helps to prevent the breakdown of body proteins. A satisfactory gastric tube feeding is that used by Bull, Joekes, and Lowe.[1] It consists of an emulsion of glucose (400 Gm.) and peanut oil (100 Gm.) in a liter of water.

Special Measures Used to Produce Diuresis.—Many procedures and drugs have been tried to produce diuresis in the oliguric and anuric period. Their

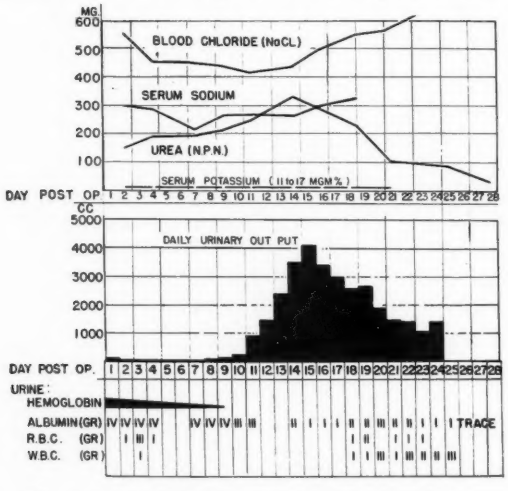

FIG. 4. Case 1. Electrolyte changes, daily urinary output, hemoglobin, and routine urinalysis findings in a case of lower nephron nephrosis.

value is not established and some of them may aggravate the condition. These include drugs such as potassium chloride, ammonium chloride, sodium sulfate, magnesium sulfate, aminophylline, alcohol-glucose solutions, histamine, sympathicolytic drugs, mercurial diuretics, and procaine intravenously. Surgical procedures have been performed in an attempt to free the kidney from neurogenic control. Favorable response to paravertebral block, spinal anesthesia, and decapsulation of the kidney have been reported. Riddle[13] stated that patients recovering from lower nephron nephrosis following decapsulation of the kidney may have recovered without the procedure since it was done when tubular regeneration was probably sufficient for survival. Roentgen therapy and diathermy to the loins have been tried, but results are not conclusive.

Substitutes for Renal Function.—Methods of substituting for renal function may be considered by the fifth or seventh day if renal failure seems to be progressing under conservative management. At present the most promising methods seem to be the artificial kidney and types of peritoneal lavage. The artificial kidney requires the constant attention of trained personnel and is not readily available to most hospitals. The dangers of heparin should be considered since it is used in the artificial kidney. Infection, obstruction of irrigating tubes by fibrin, and vomiting may make continuous peritoneal lavage difficult. Grollman, Turner, and McLean[6] recently have used intermittent peritoneal lavage on dogs as well as 5 human patients with acute renal insufficiency. Life was maintained in bilateral nephrectomized dogs for as long as sixty-nine days by this method. The gastric and intestinal mucosa have been used as dializing membranes but seem to be of more value in treating hyperpotassemia than azotemia.

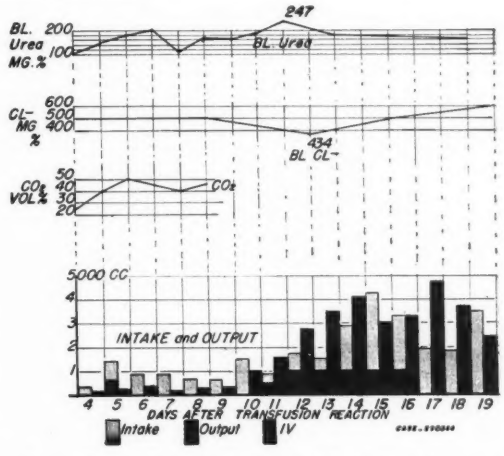

FIG. 5. Case 2. Electrolyte levels and daily intake and output of fluids in lower nephron nephrosis.

The production of diarrhea and sweating, plasmophoresis, reciprocal blood transfusions, and exsanguinotransfusions are other methods of removing waste products from the blood. The first three are not practical and may be injurious. Snapper and Schaefer[14] expressed the belief that some toxic products may be eliminated by exsanguinotransfusions that cannot be removed by dialysis.

Postanuric Phase

Attention to electrolyte and fluid balance should not be relaxed when diuresis occurs. The increased urinary output in this phase is accompanied by increased loss of sodium chloride and potassium. Dehydration may occur. After following a program of fluid and electrolyte restriction in the anuric phase

there may be a tendency to continue this treatment in the diuretic phase. Actually, large amounts of fluid and sodium chloride are necessary during diuresis. Potassium chloride intake should be adequate.

CASE REPORTS

Recently, we have had the opportunity of seeing 6 cases of lower nephron nephrosis. Probably because of the severity of their associated traumatic injuries, 3 of these patients died early after hospitalization. Another died of postoperative complications after electrolyte and fluid balance had been attained. Two of our patients survived, and the following cases are reported because of the interesting features of lower nephron nephrosis.

CASE 1.—Mrs. D. L., aged 33, was admitted to Scott and White Memorial Hospitals on March 26, 1950, for surgical removal of a herniated lumbar disk. Her general physical condition was considered to be excellent prior to surgery. Routine urine and blood studies were normal. During the surgical procedure on March 27, she was given 500 cc. of whole blood. Shortly after completion of the operation, the patient had a mild chill accompanied by cyanosis. Urine voided at that time was coffee colored and only 60 cc. in volume. This urine gave a 4 plus reaction to benzidine and guaiac. A blood sample drawn at that time revealed a level of free hemoglobin of 86 mg. per 100 cc. and a total one minute bilirubin level of 2.8 mg. per 100 cc.

During the stage of acute renal failure, the patient complained of headaches, pruritus, vertigo, and nausea. Vomiting occurred. Slight orbital edema was present but peripheral edema was absent. She was drowsy but did not lose consciousness. Temperatures the first two days after the transfusion reaction ranged from 100 to 101 F. orally. Her blood pressure reached a maximum of 164/100 during acute renal failure. Oliguria persisted for ten days and was followed by a marked diuresis. Intake and output of fluids are recorded in figure 3.

Determination of blood electrolyte levels and urine examinations are shown in figure 4. Blood urea levels (fig. 4) failed to fall until diuresis was well established. A maximum blood urea level of 330 mg. per 100 cc. was reached on the fourteenth day. Albuminuria persisted during the stage of renal failure.

From the second to the fourteenth day, intake of fluids and maintenance of nutrition was accomplished mainly with the use of gastric tube feeding recommended by Bull, Joekes, and Lowe.[1] Sodium lactate and Hartman's solution were used as aids in maintaining electrolyte balance. On the eighth day, a transfusion of whole blood was given for anemia.

Subjective symptoms gradually disappeared, and thirty-two days after the transfusion reaction, the patient was discharged from the hospital. Fifteen months after discharge, physical examination and renal function tests were normal.

CASE 2.—Mrs. W. P., aged 51, was admitted to Scott and White Memorial Hospitals on July 6, 1952, because of oliguria, weakness, and vomiting. She had received a whole blood transfusion at another hospital on July 2 for anemia. Following this transfusion she developed fever and chills. Urine passed five hours after the transfusion was described as dark red and scanty. Vomiting began the next day and persisted until admission here. She had received intravenous fluids of unknown quantity for two days prior to admission. Daily urine output before admission was estimated by the patient as 60 cc.

The patient appeared drowsy and weak. Her abdomen was distended and there was dullness to percussion in the flanks. Slight ankle, orbital, and sacral edema was present. Blood pressure on admission was 150/90. Edema present on admission gradually disappeared. The patient maintained a mild hypertension during the period of acute renal failure.

Laboratory studies on admission revealed a grade 3 albuminuria with the urine having a specific gravity of 1.010. Hemoglobin measured 10 Gm.; there were 3,800,000 red blood cells and 10,000 white blood cells; hypochromic anemia was present. Electrolyte levels during the illness and intake and output of fluids are shown in figure 5. A maximum blood urea level of 247 mg. per 100 cc. was reached on the twelfth day after the transfusion reaction. The blood chloride level (as sodium chloride) dropped to 434 mg. per 100 cc. early in the stage of diuresis, but returned to normal with increased intravenous sodium chloride administration.

While in this hospital, the patient complained of headache and anorexia, but vomiting ceased. She was able to take small quantities of a low protein, low salt diet. Oral intake was supplemented by intravenous glucose. Sodium lactate was used when the carbon dioxide combining power was low. Diuresis occurred on the tenth day following the transfusion reaction. Symptoms had subsided by the twentieth day, and the patient was dismissed from the hospital although her blood urea was still 162 mg. per 100 cc. Forty-one days after the transfusion reaction, physical examination was negative, but the blood urea was 75 mg. per 100 cc. and a twelve hour urine specimen showed a low specific gravity of 1.010.

SUMMARY

Lower nephron nephrosis occurs in a variety of conditions involving destruction of blood or tissues. Shock of various degrees is a frequent initial symptom. Oliguria or anuria is followed by acute renal insufficiency. Death is most likely to occur within the first ten days. Patients who recover develop a marked diuresis accompanied by improvement in their general clinical condition.

Conservative therapy as outlined may reduce the high mortality reported in the past. If the patient can be kept alive one or two weeks, enough tubular regeneration may be present for survival. Maintenance of electrolyte and fluid balance is stressed in the phase of acute renal failure. Dehydration, hypochloremia, hyponatremia, and hypopotassemia should be prevented in the stage of diuresis.

Case reports of 2 patients who developed lower nephron nephrosis and recovered are reviewed.

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CASE REPORTS

Bilateral Renal Cortical Necrosis in a Child Associated With an Incompatible Blood Transfusion

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BILATERAL or symmetrical renal cortical necrosis is characterized by either focal areas of coagulative necrosis or by a confluent and diffuse necrosis of the renal cortices. Since the necrosis is of the coagulative type, there is general agreement that it results from an ischemic episode. Clinically, the lesion is reflected by an acute progressive and fatal renal insufficiency. The earlier views that this lesion resulted from thrombosis or embolism have been discarded by most workers, and in recent times the ischemia has been considered to result from vasomotor disturbances within the cortex. However, there are differences of opinion as to the manner in which the vascular disturbances are initiated. Four views have been particularly prominent: (1) Scriver and Oertel[12] and Ash[1] considered the occurrence of an intense vasoconstriction followed by vasoparalysis, stasis, and thrombosis giving rise to the lesion. (2) De Navasquez[3] was unable to obtain evidence of vasoconstriction and preferred the view that loss of vasomotor tone was followed by stasis and thrombosis. (3) Trueta[14] and co-workers interpreted the disturbance as resulting from an extreme operation of their corticomedullary shunt. (4) Dunn and Montgomery,[5] supported by Heggie, suggested stasis within the capillary circulation of the glomeruli as the primary disturbance leading to a sufficient embarrassment of the blood flow to cause the overwhelming ischemia. Observations on the experimental production of this lesion by means of bacterial toxins by Smith and Muirhead[13] were considered to be consistent wtih the latter view.

With the exception of an equivocal report which will be discussed later, we have been unable to find reports in the literature of bilateral renal cortical necrosis occurring in the wake of an incompatible blood transfusion. The first case reported here is considered of interest in view of a clear-cut association with an incompatible blood transfusion and the development of the lesion in a 7 year old child. The second case, presented briefly, emphasizes the more common clinical background in the development of this lesion, namely, in association with a complication of pregnancy.

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CASE REPORTS

CASE 1.—A 7 year old Negro girl entered the hospital twenty-four hours after incurring second and third degree burns over the lower part of the abdomen, the entire perineum, and the anterior portions of the thighs. The burns were estimated to cover 25 to 30 per cent of the body surface. The blood pressure on admission was 70/60 mm. of mercury, pulse 150 per minute, respirations 24 per minute, and temperature 102 F. The initial laboratory data revealed a hemoglobin of 14.2 Gm. per 100 cc. of blood, and a leukocytosis of 21,000 white cells per cubic millimeter of blood with a moderate left shift. The administration of 2 L. of 5 per cent dextrose in Ringer's lactate solution and 500 cc. of 10 per cent dextrose in distilled water was followed by a drop in the hemoglobin concentration to 9.5 Gm. per 100 cc., the red blood cell count to 3,200,000 by the end of forty-eight hours. The patient was able to tolerate oral feedings well. No further fluids were administered intravenously until the thirty-fourth hospital day.

The patient was of blood type AB, Rh (D) negative. Shortly after admission, she received 500 cc. of this type of blood, but all subsequent transfusions entailed the adminis-

tration of type O, Rh (D) negative blood to which had been added types specific A and B substance, as available on the market.

Until the thirty-third hospital day, the patient progressed favorably with supportive treatment which included sulfasuxidine by mouth, analgesics, salt solution by mouth, debridement, and razor grafts of the burns under general anesthesia. By the thirty-third hospital day, the patient had received 2,000 cc. of whole blood, 500 cc. of type AB blood previously mentioned, 1,000 cc. of type O, Rh negative blood with A and B substance added, and 500 cc. of type O, Rh negative blood without A and B substance added. On this day, after the administration of 150 cc. of type O, Rh (D) negative blood without A and B substance, the patient developed a reaction which was characterized by a sudden chill and fever with the temperature being elevated to 103 following eight days, the blood urea level rose progressively, reaching a maximum of 535 mg. per 100 cc., and the hemoglobin receded to 6.3 Gm. per 100 cc. The patient received another transfusion of whole blood of 500 cc. without a reaction. She remained lucid for ten days. Suddenly on the eleventh day after the transfusion reaction, there was an episode of respiratory distress and peripheral circulatory failure, followed within a few minutes by death, the respirations ceasing first (table 1).

Autopsy Findings.—Postmortem blood culture six and one-half hours after death revealed Proteus vulgaris (a probable contaminant). Slight bronchopneumonia was present. Together the kidneys weighed 189 Gm. The capsule stripped with ease revealing a diffuse, yellow discoloration of the surface. On cross section, almost the entire cortex was yellow and soft. The yellow zone was surrounded by a thin border of hyperemia. There were focal areas of normal appearing cortex. Microscopic examination revealed slight sparing of

TABLE 1.—*Laboratory Data of Case 1 Following the Blood Transfusion Reaction.*

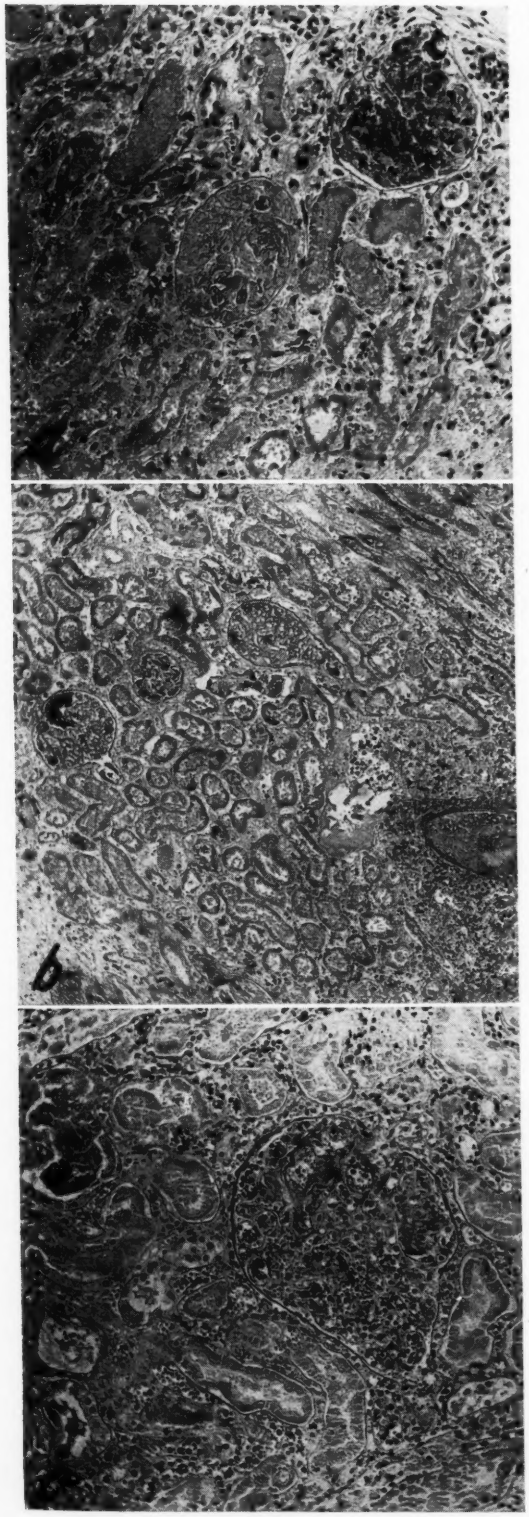
Hospital Day	Oral Intake (cc.)	Urine Output	Intravenous Fluid	Whole Blood	Hemoglobin (Gm.)	Urea (mg./100 cc.)	Miscellaneous
33	1,550	5+ cc.		150 cc. (reaction)			
34	1,630	0	1,000 cc. dextrose 500 cc. salt		11.5		
35	375	0			8.7	150	
36	150 H_2O 150 milk	7 cc.			7.5	197	
37	100 H_2O 200 fat	0				298	CO_2 29.8 mEq/L.
38	150 salt 100 fat 200 H_2O	0	100 cc. Ringer's lactate	250 cc.		342	CO_2 21.5 mEq/L.
39	200 H_2O 200 salt 200 fat	0					
40	200 H_2O 200 fat	0	100 cc. Ringer's lactate	250 cc.		496	
41	500 H_2O 100 salt 200 fat	0			6.3	449	CO_2 22.6 mEq/L. Cl 89.4 mEq/L.
42	500 H_2O 200 fat 150 salt	0				535	
43	500 H_2O 200 fat 300 salt	0			6.3		
44	300 H_2O 300 salt 200 fat	0				471	CO_2 18.5 mEq/L. Cl 93.3 mEq/L.
45							Died

F., receding within eight hours to 95 F. Hypothermia persisted for eight hours, following which the temperature returned to normal. At the time of the reaction, there was abdominal pain, vomiting, diarrhea, and tachycardia, but no significant change in the blood pressure. The urine specimens collected after the reaction were wine colored. The following day, the patient was jaundiced and developed anuria. Upon recrossmatching the donor cells, the blood was found to be compatible by the routine procedures. A crossmatch of the donor's cells with the patient's serum by the indirect Coombs test later was found to be compatible.

By the thirty-fifth hospital day, the blood urea level was 150 mg. per 100 cc., the icterus index 36, creatinine 4.5 mg. per 100 cc. On the thirty-sixth hospital day, the patient excreted 7 cc. of urine which was found to yield a 3 plus albumin test, many white blood cells, and an occasional red blood cell. At this time, the blood urea measured 197 mg. per 100 cc., creatinine 4.5 mg., uric acid 4.5 mg. During the

the renal tubules and an occasional intact glomerulus at the extreme periphery of the cortex just beneath the capsule. The majority of the cortex was involved in a coagulative necrosis which included approximately 75 to 80 per cent of the thickness of the cortex with sparing of the renal cortical parenchyma at the juxtamedullary zone. The glomeruli within the necrotic cortex were congested. There were multiple foci of calcification involving the damaged glomeruli and tubules. In some areas, the calcification had completely replaced glomeruli. The glomeruli and tubules within the juxtamedullary zone were essentially unaffected, although many heme casts were present within the lumen of these tubules. The medulla was intact (fig. 1a and b).

Comment.—This patient had an incompatible blood transfusion on the thirty-third hospital day. This appears definite since there was the typical acute reac-



tion characterized by hemoglobinuria followed by jaundice and renal failure. It was not demonstrated objectively, however, whether the hemolysis resulted from anti-A and anti-B substances in the type O blood used or whether it was due to some unusual incompatibility involving the donor's cells. Against the latter view was the demonstration of a negative indirect Coombs test with the donor's cells and the patient's serum on the sixth day. In favor of the former view was the subsequent rapid development of a more pronounced anemia in the absence of blood loss. There is little doubt also that the renal insufficiency began immediately following this episode. The renal insufficiency was of the acute, progressive, unrelenting type, and its termination was similar to that observed in other cases with this form of acute renal failure.[11] The unrelenting uremia in this case, however, resulted from bilateral cortical necrosis as demonstrated by the autopsy. All of these observations suggest that the renal cortical necrosis was associated with the reaction due to incompatible blood.

CASE 2.—A 32 year old Negro woman, gravida 7, para 5, was admitted to the hospital with the history of vaginal bleeding for two days, chills and fever, generalized malaise, and a purple rash. On the day of admission, the patient passed a fetus described as compatible with six months of gestation. Physical examination revealed a temperature of 102 F., pulse 120 per minute, respirations 60 per minute, blood pressure 56/40 mm. of mercury. Petechial hemorrhages were scattered over the body; there was slight jaundice of the sclerae; and the patient was unable to distinguish light from dark. The lungs were clear; the bowel sounds were normal. The laboratory data indicated red blood cells 3,900,000, hemoglobin 9.5 Gm. per 100 cc., white blood cells 55,000 with 3 per cent myelocytes, 7 per cent young cells, 32 per cent bands, 50 per cent segmented forms, and 8 per cent lymphocytes. The blood culture yielded an unidentified gram negative bacillus. The patient had hallucinations, became delusional, and rapidly progressed downhill, dying five and one-half hours after admission.

Autopsy Findings.—There were multiple purpuric and ecchymotic hemorrhages over the skin and tongue. There was generalized pulmonary edema and acute interstitial myocarditis. The uterus was enlarged and demonstrated necrotizing and gangrenous endometritis and myometritis. Both adrenal glands were hemorrhagic and microscopically demonstrated diffuse infarction. The kidneys were enlarged and heavy. The right weighed 225 Gm. and the left 220 Gm. On stripping the capsule, the surface appeared mottled. On section, the parenchyma bulged. There were multiple hemorrhagic areas scattered throughout the entire cortex and multiple grayish areas measuring 0.5 to 1.0 cm. in greatest dimension, extending from the pyramids to the capsule. An

FIG. 1a. Case 1. Photomicrograph showing marked coagulative necrosis of the renal cortex and the somewhat unusual finding of focal calcification. x 135.

b. Case 1. A lower magnification showing focal thrombi within the glomeruli. x 60.

c. Case 2. Photomicrograph showing earlier changes of renal cortical necrosis. The most outstanding changes illustrated are severe congestion and fibrin thrombi of the glomerular capillaries, early tubular necrosis, and albumin casts. x 131.

estimated 50 to 75 per cent of the cortex was involved in this process of infarction. Microscopically, the kidneys revealed findings which indicated bilateral renal cortical necrosis. The necrosis extended all the way from the capsule to the medulla, but did not involve the latter. The capillaries in the glomeruli were filled with reddish thrombi, some of which extended into the arterioles. There was necrosis of the arterioles. In focal areas, the glomeruli and the peritubular capillaries were markedly engorged with red blood cells. Moderate infiltration with polymorphonuclear neutrophilic leukocytes was observed. Hyaline casts were abundant in the tubules throughout the areas of involvement. The medulla was intact (fig. 1c).

Comment.—This case demonstrated a typical, fulminant, septic abortion with the added feature of bilateral renal cortical necrosis. The necrosis was in its developmental phase but clear-cut in its gross and microscopic appearance. A similar case with bilateral adrenal infarction has been reported.[9] Bilateral cortical necrosis with associated adrenal infarction has been produced experimentally.[2]

DISCUSSION

Since the report of Duff and More[4] in 1941, our search indicates that 78 additional cases of bilateral renal cortical necrosis have been reported in the literature, including our 2 cases, making the total 149 cases reported since the original description by Juhel-Rénoy[6] in 1886. Of the 78 cases, 38 were associated with pregnancy, and 40 were associated with other causes. Tables 2 and 3 record a breakdown of the clinical background associated with bilateral renal cortical necrosis.

The cases associated with pregnancy (total of 86) may be divided into four main categories: (1) those associated with abruptio placenta, with or without other signs of toxemia of pregnancy, constituting 59 per cent of the total; (2) those associated with signs and symptoms of toxemia, including convulsive and nonconvulsive toxemia with or without accidental hemorrhage, which constitute 23 per cent of the total; (3) those associated with abortions, 6 per cent of the total, half of which were septic; and finally, (4) a miscellaneous group including a series of isolated complications of pregnancy (12 per cent). These 86 cases followed the pattern previously emphasized by Duff and More[4] wherein 56 per cent of the patients were over the age of 30, and more than 60 per cent occurred in the last trimester of pregnancy.

MacGillivray,[7, 8] in reviewing the cases of bilateral renal cortical necrosis associated with pregnancy appearing in the British literature, concluded that toxemia or sepsis with an incidental shock represented the main clinical background which was conducive to the production of the cortical necrosis. Of the cases in nonpregnant patients, there has been a fairly equal distribution between males and females, and 20 out of 63 cases have occurred in the pediatric age group. As listed in table 3, the main background for renal cortical necrosis in this group has been infection and various forms of intoxications, all of which were frequently associated with shock. Thus, despite the need to separate the clinical background into a pregnant and nonpregnant group, there nevertheless are similarities between the two in the sense of a common encounter of infection, intoxication, and peripheral circulatory failure.

It is of interest that the background of infection, intoxication, and shock is also conducive to the development of the recoverable type of acute renal failure which has been designated as "lower nephron nephrosis." In tables 2 and 3 an attempt has been made to list the various types of clinical background associated with development of bilateral renal cortical necrosis and "lower nephron nephrosis." The groupings were obtained from the 149 cases of renal cortical necrosis available in the literature and the first 296 cases of acute renal insufficiency of the "lower nephron nephrosis" type, culled without selection out of the recent literature on this subject. It is evident from tables 2 and 3 that there is a considerable overlapping in the clinical background associated with both development of renal cortical necrosis and "lower nephron nephrosis." The one common factor between these two conditions that is outstanding is that of peripheral circulatory failure. In general, both conditions occur in association with trauma, infections, and various types of intoxications, usually associated with overt peripheral circulatory failure. However, within isolated backgrounds, there are outstanding differences in the incidence of these two types of renal injuries. Particularly outstanding is the high incidence of the recoverable type of acute renal failure designated as "lower nephron nephrosis" as a consequence of trauma, incompatible blood transfusions, certain intoxications such as sulfonamide, carbon tetrachloride, and mercury poisoning, and posthemorrhagic shock; for bilateral cortical necrosis, abruptio placenta, various types of toxemia of pregnancy, and severe infections are particularly outstanding backgrounds. This overall overlapping in background can be interpreted as indicating a possibility of common factors in the pathogenesis of these two lesions. The outstanding differences between certain backgrounds, on the other hand, suggest additional fundamental differences in the pathogenesis.

Since most conditions that give rise to bilateral renal cortical necrosis appear to be eligible at other times to initiate the recoverable type of renal insufficiency, and vice versa, it is of interest that reactions due to incompatible blood transfusions, a disturbance that frequently causes renal insufficiency of the recoverable type, has not been considered a causative

background for bilateral renal cortical necrosis. In the literature we have encountered only 1 case[15] in which an incompatible transfusion might be considered related to cortical necrosis, and the case was inconclusive in that the patient had received an absorbable sulfonamide at the time of the development of the renal injury. The case herein reported appears to be a clear-cut incident of a hemolytic transfusion reaction followed by the development of unrelenting renal failure due to bilateral cortical necrosis. Incompatible transfusion reaction is a condition which, when associated with hemoglobinuria as in the present case, in our experience[10] is followed in 90 per cent of the cases with some degree of renal insufficiency, but which rarely is associated with cortical necrosis. It is conceivable that this indicates again overlapping factors in the pathogenesis of both of these lesions, but that in each instance additional factors are needed to complete the pathogenesis. The occurrence of the additional factors in incompatible blood transfusions necessary for renal cortical necrosis appears to be rare.

The clinical course of the acute, progressive, unrelenting failure due to a widespread tubular nephropathy, as previously described,[11] is similar to that associated with bilateral renal cortical necrosis as exemplified by the cases herein reported. We have been unable to ascertain a clinical means of differentiating these two forms of overwhelming renal injury. The ultimate differentiation at present rests on the morphologic study at autopsy.

TABLE 2.—*Reported Cases of Bilateral Renal Cortical Necrosis and "Lower Nephron Nephrosis" Associated with Pregnancy.*

Clinical Background	Bilateral Renal Cortical Necrosis (No. Cases)	Lower Nephron Nephrosis (No. Cases)
Abruptio placenta and preeclampsia or eclampsia*	28	6
Abruptio placenta (alone)	23	0
Preeclampsia, eclampsia, or other toxemia	20	4
Septic abortion	3	1
Abortion or postabortion	2	10
Miscellaneous		
Postpartum hemorrhage	2	2
Placenta previa	1	0
Thrombosis of ovarian and renal veins	1	0
Shock during cesarean section	1	0
Urinary tract infection	1	0
Ruptured dissecting aneurysm	1	0
Sulfonamide and questionable transfusion reaction	1	0
Sulfonamide intoxication	1	0
None given	1	0
Totals	86	23

*Eclampsia defined as eclamptic convulsive seizures.

CONCLUSIONS

Two cases of bilateral renal cortical necrosis displaying the syndrome of acute, progressive, unrelenting renal failure have been presented. One case was unique in that the renal lesion followed in the wake of an incompatible and hemolytic blood transfusion reaction. The second case followed the classical pattern associated with a complication of pregnancy.

Although incompatible blood transfusions associated with hemoglobinuria frequently are associated with the development of acute renal failure, the presently reported case appears to be a rare demonstration of the relationship of an incompatible transfusion and bilateral renal cortical necrosis.

The clinical background for the development of bilateral renal cortical necrosis and the recoverable type of acute renal failure designated as "lower nephron nephrosis" has been summarized. From this summary, it has been emphasized that marked overlapping occurs in the background of both of these conditions, but that the incidence of each lesion is more closely aligned with certain types of backgrounds than with others. This type of overlapping suggests similarities and dissimilarities in the factors concerned with the pathogenesis of the two lesions.

TABLE 3.—*Reported Cases of Bilateral Renal Cortical Necrosis and "Lower Nephron Nephrosis" Not Associated with Pregnancy.*

Clinical Background	Bilateral Renal Cortical Necrosis (No. Cases)	Lower Nephron Nephrosis (No. Cases)
Infection	25	5
Trauma	3	148
Incompatible blood transfusion	1	50
Erythroblastosis fetalis	1	0
Transurethral resection	0	6
Poisons and drugs		
Sulfonamides	2	12
Carbon tetrachloride	0	11
Mercury bichloride	0	6
Potassium permanganate and mercury bichloride	0	1
Local anesthesia	1	0
Iodine	1	0
Epinephrine	1	0
Cobra venom	1	0
Camphor	1	0
Almond extract	1	0
Pentachlornephthalene	0	1
Bacitracin	0	1
Acute or chronic alcoholism	4	3
Operative, postoperative, and hemorrhagic shock	4	21
Vomiting and/or diarrhea	8	4
Miscellaneous		
Periarteritis nodosa	1	0
Anoxia (prolonged labor)	1	0
Tachycardia and shock	0	1
Bilateral ureteropelvic obstruction	0	1
Diabetic acidosis with shock	0	1
Thrombocytopenic purpura	1	0
Hepatorenal syndrome	0	1
Hypertension and syphilis	1	0
Hypertension, multiple splenic infarcts	1	0
Semicoma, dyspnea (progressive)	1	0
General malaise, backache, and hypothermia	1	0
No overt disease	2	0
Totals	63	273

Incompatible blood transfusions with hemoglobinuria seem to belong in the category frequently associated with the recoverable type of acute renal failure

and infrequently associated with the development of bilateral renal cortical necrosis.

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15. Werner, W. A.; Muckerman, R. I. C.; and Allen, H. N.: Bilateral Cortical Necrosis of Kidneys, J. Missouri M. A. *47:*20-22 (Jan.) 1950.

ABSTRACT OF DISCUSSION

DR. E. E. MUIRHEAD, Dallas: This case report and general discussion dealing with the development of bilateral renal cortical necrosis in the wake of an incompatible blood transfusion brings to light the entire problem of acute progressive unrelenting renal failure. By this term is meant the occurrence of sudden acute renal failure with no signs of recovery, with fixed persistent, or at best slightly improved, oliguria, with marked azotemia, prominent acidosis, and a tendency toward prominent hyponatremia and hypochloremia, followed in time by hypertension and the possibility of mental disturbances, diarrhea, and hyperkalemia, the entire complex ending fatally in from ten to more than twenty-one days.

This syndrome of acute progressive unrelenting renal failure results from a series of different injuries to the kidney, most of which appear to be mediated through a sudden vascular disruption of the kidney. Thus, similar clinical manifestations ending in fatal uremia may arise from the following conditions: bilateral renal cortical necrosis, bilateral renal papillary necrosis, bilateral renal artery obstruction, bilateral multiple infarcts of the kidneys, bilateral renal vein thrombosis in some instances, collagen disease with overwhelming arteritis (particularly periarteritis nodosa), malignant hypertension with widespread vascular disruption, and finally, overwhelming acute tubular nephropathy of the type following peripheral circulatory failure or nephrotoxic injury to the kidney. All of these conditions have features that resemble the state of bilateral nephrectomy induced in experimental animals and apparently represent overwhelming destruction of renal substance. It is of interest that the acute tubular nephropathy which has been given different names such as "lower nephron nephrosis," the "anoxic kidney," "hemoglobinuric nephrosis," and "tubular nephritis" on occasions is associated with acute progressive unrelenting renal failure. In my experience approximately 10 per cent of patients with this tubular disturbance die with unrelenting renal failure. Under these conditions the tubular injury is not only confined to one segment of the tubule but involves the entire nephron from the glomerulus to the collecting segment. It is frequently exceedingly difficult to differentiate clinically between this type of overwhelming tubular nephropathy and bilateral renal cortical necrosis as described in the preceding report. Indeed, at times one has to rely on the autopsy findings for this separation. Since these various injuries to the kidney evoke similar clinical manifestations, however, this is mainly an academic point.

NEWS

COMING MEETINGS AND CLINICS

Texas Medical Association, San Antonio, May 3-5, 1954. Dr. George Turner, El Paso, Pres.; Mr. N. C. Forrester, 1801 Lamar Blvd., Austin, Executive Secy.

American Medical Association, St. Louis, Dec. 1-4, 1953. Dr. Edward J. McCormick, Toledo, Ohio, Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy, Houston, Feb. 1-3, 1954. Dr. Ben Z. Rappaport, Chicago, Pres.; Dr. Alan G. Cazort, 1425 West 7th St., Little Rock, Secy.

American Academy of Dermatology and Syphilology, Chicago, Dec. 5-10, 1953. Dr. Michael H. Ebert, Chicago, Pres.; Dr. John E. Rauschkolb, 25 Prospect Ave., N W., Cleveland 15, Secy.

American Academy of General Practice. Dr. R. B. Robins, Camden, Ark., Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Secy.

American Academy of Obstetrics and Gynecology, Cincinnati, Dec. 14-16, 1953. Dr. Robert A. Kimbrough, Jr., Philadelphia, Pres.; Dr. C. Paul Hodgkinson, Detroit, Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 12-16, 1953. Dr. Frederick C. Cordes, San Francisco, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Miami, Oct. 6-9, 1953. Dr. Phillip Barba, Philadelphia, Pres.; Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, Montreal, Canada, May 3-5, 1954. Dr. Emile F. Holman, San Francisco, Pres.; Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif., Secy.

American Association of Genito-Urinary Surgeons, Shawnee-on Delaware, Pa., May 26-28, 1954. Dr. Vincent J. O'Conor, Chicago, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons. Dr. Nicholson J. Eastman, Baltimore, Pres.; Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Secy.

American Cancer Society. Dr. Guy Aud, Louisville, Ky., Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Executive Vice-Pres.

American College of Allergists, Miami Beach, April 5-10, 1954. Dr. M. Murray Peshkin, New York, Pres.; Dr. Fred W. Wittich, 423 La Salle Medical Bldg., Minneapolis 2, Secy.

American College of Chest Physicians, San Francisco, June 17-20, 1954. Dr. Alvis E. Greer, Houston, Pres.; Mr. Murray Kornfeld, 112 E. Chestnut St., Chicago 11, Executive Secy.

American College of Physicians, Chicago, April 5-9, 1954. Dr. Leroy H. Sloan, Chicago, Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology, Chicago, February, 1954. Dr. John D. Camp, Los Angeles, Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons, Chicago, Oct. 5-9, 1953. Dr. Harold L. Foss, Danville, Pa., Pres.; Dr. Michael L. Mason, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine and Rehabilitation. Dr. Walter M. Solomon, Cleveland, Pres.; Dr. Frances Baker, 1 Tilton Ave., San Mateo, Calif., Secy.

American Dermatological Association, White Sulphur Springs, W. Va., April 13-17, 1954. Dr. Louis A. Brunsting, Rochester, Minn., Pres.; Dr. J. Lamar Callaway, Duke Hospital, Durham, N. C., Secy.

American Gastro-Enterological Association, San Francisco, June 18-19, 1954. Dr. J. M. Ruffin, Durham, N. C., Pres.; Dr. H. Marvin Pollard, 1313 E. Ann St., Ann Arbor, Mich., Secy.

American Gynecological Society, Hot Springs, Va., May 20-22, 1954. Dr. Richard W. TeLinde, Baltimore, Pres.; Dr. John I. Brewer, 104 S. Michigan Ave., Chicago, Secy.

American Heart Association, Chicago, April 1-4, 1954. Dr. Robert L. King, Seattle, Pres.; Dr. William H. Bunn, 44 E. 23d St., New York 10, Secy.

American Hospital Association. Dr. Edwin L. Crosby, Chicago, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, Boston, May 25-27, 1954. Dr. LeRoy A. Schall, Boston, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 7, N. Y., Secy.

American Neurological Association, Atlantic City, N. J., June 14-16, 1954. Dr. Roland P. Mackay, Chicago, Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, Glacier Park, Mont., June 16-18, 1954. Dr. William L. Benedict, Rochester, Minn., Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, Bretton Woods, N. H., June 6-9, 1954. Dr. A. R. Shands, Jr., Wilmington, Del., Pres.; Dr. George O. Eaton, 4 E. Madison St., Baltimore 2, Secy.

American Pediatric Society, Buck Hill Falls, Pa., May 3-5, 1954. Dr. Rustin McIntosh, New York, Pres.; Dr. Aims McGuinness, 237 Medical Laboratory, University of Pennsylvania, Philadelphia 46, Secy.

American Proctologic Society, Los Angeles, June 2-5, 1954. Dr. Wendell Green, Toledo, Ohio, Pres.; Dr. Stuart T. Ross, 131 Fulton Ave., Hempstead, N. Y., Secy.

American Psychiatric Association, St. Louis, May 3-7, 1954. Dr. Kenneth E. Appel, Philadelphia, Pres.; Dr. R. Finley Gayle, 6300 Three Chopt Rd., Richmond 21, Va., Secy.

American Public Health Association, New York, Nov. 9-13, 1953. Dr. Wilton L. Halverson, San Francisco, Pres.; Dr. R. W. Atwater, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists, Seattle, Oct. 6-9, 1953. Dr. Ralph T. Knight, Minneapolis, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists, Chicago, Oct. 12-16, 1953. Dr. Henry F. Hunt, Danville, Pa., Pres.; Dr. Clyde G. Culberson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association, Cleveland, April, 1954. Dr. Howard C. Naffziger, San Francisco, Pres.; Dr. R. Kennedy Gilchrist, 59 East Madison St., Chicago, Secy.

American Urological Association, New York, May 31-June 3, 1954. Dr. J. A. Campbell Colston, Baltimore, Md., Pres.; Dr. C. H. DeT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons, Chicago, Oct. 8-10, 1953. Dr. Charles L. Farrell, Pawtucket, R. I., Pres.; Mr. Harry E. Northam, 360 N. Michigan Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter. Dr. William R. Lovelace, Albuquerque, N. M., Pres.; Dr. Karl Meyer, 1516 Lake Shore Drive, Chicago, Secy.

National Tuberculosis Association, Atlantic City, N. J., May 17-21, 1954. Mr. Mark H. Harrington, Denver, Pres.; Mr. Kemp D. Battle, 1790 Broadway, New York 19, Secy.

Radiological Society of North America, Chicago, Dec. 13-18, 1953. Dr. Ira H. Lockwood, Kansas City, Mo., Pres.; Dr. D. S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

Southern Medical Association, Atlanta, Ga., Oct. 26-29, 1953. Dr. Walter C. Jones, Miami, Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham 3, Ala., Secy.

Southern Psychiatric Association, Biloxi, Miss., Oct. 4-6, 1953. Dr. R. Burke Suitt, Durham, N. C., Pres.; Dr. Joseph L. Knapp, 210 N. Westmoreland, Dallas, Secy.

Southern Surgical Association, Hot Springs, Va., Dec. 8-10, 1953. Dr. I. A. Bigger, Richmond, Va., Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville 5, Tenn., Secy.

Southwest Allergy Forum. Dr. Orval R. Withers, Kansas City, Mo., Pres.; Dr. Frederic Speer, 2601 Parallel Ave., Kansas City 4, Mo., Secy.

Southwest Regional Cancer Conference. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association, Tucson, Oct. 29-31, 1953. Dr. Wesley O. Connor, Jr., Albuquerque, N. M., Pres.; Dr. Celso Stapp, 800 Montana, El Paso, Secy.

Southwestern Surgical Congress. Dr. Louis P. Good, Texarkana, Ark., Pres.; Dr. C. R. Rountree, 1227 Classen, Oklahoma City 3, Secy.

Tri-State Medical Assembly. Dr. Frank G. Thibault, El Dorado, Ark., Pres.; Dr. Albert M. Hand, Le Bonheur Children's Hospital, Memphis 3, Tenn., Secy.

United States-Mexico Border Public Health Association, Albuquerque, N. M., April, 1954. Mr. Richard F. Poston, Dallas, Pres.; Dr. Sidney B. Clark, 314 U. S. Court House, El Paso, Secy.

STATE

Private Clinics and Hospitals Association of Texas, Austin, Dec. 5-6, 1953. Dr. S. D. Coleman, Navasota, Pres.; Dr. Neil D. Buie, Marlin, Secy.

Texas Academy of General Practice. Dr. C. U. Callan, Rotan, Pres.; Dr. Woodson W. Harris, 1410 Nickerson, Austin, Secy.

Texas Academy of Internal Medicine, Houston, Dec. 5-6, 1953. Dr. Hatch W. Cummings, Jr., Houston, Pres.; Dr. George M. Jones, 1314 Medical Arts Bldg., Dallas, Secy. Meetings restricted to members.

Texas Air-Medics Association, San Antonio, May, 1954. Dr. George L. Gallaher, Harlingen, Pres.; Dr. J. S. Minnett, 2512 Welborn, Dallas, Secy.

Texas Association of Blood Banks, Fort Worth, Dec. 4-5, 1953. Dr. W. N. Powell, Temple, Pres.; Miss Majorie Saunders, 3500 Gaston Ave., Dallas, Secy.

Texas Association of Obstetricians and Gynecologists, Waco, Feb. 19-20, 1954. Dr. D. D. Wall, San Angelo, Pres.; Dr. Carey Hiett, 815 Fifth Ave., Fort Worth, Secy.

Texas Chapter, American College of Chest Physicians, San Antonio, May 2, 1954. Dr. Henry H. Hoskins, San Antonio, Pres.; Dr. Walter C. Brown, 1733 S. Brownlee Blvd., Corpus Christi, Secy.

Texas Club of Internists. Dr. Joseph F. McVeigh, Fort Worth, Pres.; Dr. Charles Darnall, Capital National Bank Bldg., Austin, Secy.

Texas Dermatological Society, San Antonio, May 2, 1954. Dr. Maurice Barnes, Waco, Pres.; Dr. Thomas L. Shields, 1216 Pennsylvania Ave., Fort Worth, Secy.

Texas Diabetes Association, San Antonio, May 2, 1954. Dr. Raymond L. Gregory, Galveston, Pres.; Dr. Edmond K. Doak, 501 Hermann Professional Bldg., Houston, Secy.

Texas Division, American Cancer Society, Austin, Jan. 14-15, 1954. Mr. Travis Wallace, Dallas, Pres.; Mr. J. Louis Neff, 1609 Colorado, Austin, Executive Director.

Texas Heart Association, San Antonio, May 3, 1954. Dr. Joseph McVeigh, Fort Worth, Pres.; Miss Roberta Miller, 412 Fidelity Union Life Bldg., 1511 Bryan St., Dallas, Executive Secy.

Texas Hospital Association, Houston, May 18-20, 1954. Mr. W. U. Paul, El Paso, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association, Mexico City, Nov. 6-7, 1953. Dr. James Blair, San Antonio, Pres.; Dr. John L. Otto, John Sealy Hospital, Galveston, Secy.

Texas Orthopedic Association, San Antonio, May 3, 1954. Dr. Edward T. Smith, Houston, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Corpus Christi, Oct. 2-3, 1953. Dr. J. A. Bybee, Beaumont, Pres.; Dr. M. C. Carlisle, 1410 Austin Ave., Waco, Secy.

Texas Public Health Association, Galveston, Feb. 14-17, 1954. Dr. Roy G. Reed, La Marque, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.

Texas Radiological Society, Dallas, Jan. 29-30, 1954. Dr. J. E. Miller, Dallas, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association, San Antonio, May 3, 1954. Dr. William E. Crump, Wichita Falls, Pres.; Dr. W. D. Marrs, 306 Broadway, Fort Worth, Secy.

Texas Rheumatism Association, Houston, Dec. 4, 1953. Dr. J. C. Crager, Beaumont, Pres.; Dr. Charles H. Cornwell, Marlin, Secy.

Texas Society for Mental Health, Waco, March 4-6, 1954. Mrs. Frank Schoonover, Fort Worth, Pres.; Mrs. Elizabeth F. Gardner, 2504 Jarratt Ave., Austin 21, Executive Secy.

Texas Society of Anesthesiologists, San Antonio, May 2, 1954. Dr. James B. Robinett, Jr., Houston, Pres.; Dr. C. R. Allen, John Sealy Hospital, Galveston, Secy.

Texas Society of Gastroenterologists and Proctologists, San Antonio, May, 1954. Dr. Cecil O. Patterson, Dallas, Pres.; Dr. W. T. Arnold, 1402 Hermann Prof. Bldg., Houston, Secy.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1953. Dr. Dan Brannin, Dallas, Pres.; Dr. Lyle Hooker, 627 Esperson Bldg., Houston, Secy.

Texas Society of Pathologists, Galveston, Jan. 31, 1954. Dr. A. O. Severance, San Antonio, Pres.; Dr. Lloyd R. Hershberger, Shannon Memorial Hospital, San Angelo, Secy.

Texas Surgical Society, Dallas, Oct. 5-6, 1953. Dr. George W. Waldron, Houston, Pres.; Dr. Albert W. Hartman, 414 Navarro St., San Antonio 5, Secy.

Texas Tuberculosis Association, Dallas, April 9-10, 1954. Dr. R. G. McCorkle, San Antonio, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.

Texas Urological Society. Dr. R. E. Cone, Galveston, Pres.; Dr. Ray M. Morgan, Jr., Hermann Hospital, Houston, Secy.

DISTRICT

First District Society, Pecos, February, 1954. Dr. John W. O'Donnell, Alpine, Pres.; Dr. H. D. Garrett, First National Bldg., El Paso, Secy.

Second District Society, Sweetwater, April 15, 1954. Dr. T. D. Young, Sweetwater, Pres.; Dr. Francis Hood, Sweetwater, Secy.

Third District Society, Amarillo, April 13, 1954. Dr. Robert A. Neblett, Canyon, Pres.; Dr. James T. Hall, 1626 Fifteenth St., Lubbock, Secy.

Fourth District Society, Brownwood, Oct. 8, 1953. Dr. Lloyd R. Hershberger, San Angelo, Pres.; Dr. Paul M. Wheelis, Brownwood, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July, 1954. Dr. Y. C. Smith, Sr., Corpus Christi, Pres.; Dr. Robert J. Sigler, 1126 Third Street, Corpus Christi, Secy.

Seventh District Society, Austin, Feb. 18, 1954. Dr. Benjamin Clary Bates, Austin, Pres.; Dr. William McLean, 4618 Burnet Road, Austin, Secy.

Eighth District Society, Wharton, Oct. 8, 1953. Dr. Andrew S. Tomb, Victoria, Pres.; Dr. George E. Glover, Jr., Victoria, Secy.

Ninth District Society, Brenham, March 25, 1954. Dr. George D. Bruce, Baytown, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, Secy.

Tenth District Medical Society, Port Arthur, Oct. 3, 1953. Dr. Edmund D. Jones, Beaumont, Pres.; Dr. O. W. Harris, Jr., Liberty, Secy.

Eleventh District Society, Henderson, Oct. 29, 1953. Dr. Melvin R. Wilcox, Athens, Pres.; Dr. Marlin T. Braswell, Henderson, Secy.

Twelfth District Society, Waco, January, 1954. Dr. Howard Dudgeon, Jr., Waco, Pres.; Dr. Walter B. King, Jr., Waco, Secy.

Thirteenth District Society, Fort Worth, 1954. Dr. William E. Crump, Wichita Falls, Pres.; Dr. Robert D. Moreton, 815 Medical Arts Bldg., Fort Worth, Secy.

Fourteenth District Society, Denton, 1954. Dr. J. W. Atchison, Gainesville, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.

Fifteenth District Society, Longview, April 20, 1954. Dr. L. E. Rutledge, Daingerfield, Pres.; Dr. George Tate, Longview, Secy.

CLINICS

Dallas Southern Clinical Society, Dallas, March 15-18, 1954. Miss Helga Boyd, Medical Arts Bldg., Dallas 1, Executive Secy.

Central Texas Spring Clinic, Waco, April 7, 1954. Dr. Walter B. King, 2320 Columbus Ave., Waco, Secy.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 26-28, 1954. Dr. John M. Smith, Jr., 205 Camden St., San Antonio, Secy.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1954. Dr. Maurice E. St. Martin, Room 103, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference. Dr. C. H. Wilson, Wichita Falls, Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 26-29, 1953. Miss Alma F. O'Donnell, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Postgraduate Medical Assembly of South Texas, 1954. Dr. Edward T. Smith, 906 Hermann Professional Bldg., Houston, Secy.

State Tumor Conference, Wichita Falls, March 31, 1954. Dr. Bailey R. Collins, 925½ Scott Street, Wichita Falls, Director.

BOARD EXAMINATIONS

Texas State Board of Examiners in Basic Sciences, Austin, Oct. 16-17, 1953. Mrs. Betty Ratcliff, 407 Perry-Brooks Bldg., Austin, Chief Clerk.

Texas State Board of Medical Examiners, Fort Worth, Nov. 12-14, 1953. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Secy.

CENTRAL ASSOCIATION OF OBSTETRICIANS

Members of the medical profession in Texas are invited to participate in the convention of the Central Association of Obstetricians and Gynecologists to be held November 5-7 at the Shamrock Hotel in Houston, the secretary, Dr. Harold L. Gainey, Kansas City, states. No registration fee will be charged.

Afternoon scientific programs the first two days will be presented by members of the Houston Obstetrical and Gynecological Society. Other speakers tentatively scheduled include Drs. David A. Bickel, South Bend, Ind.; Paul D. Bruns, Denver; Jason H. Collins, New Orleans; Eduard Eichner, Cleveland; William F. Guerriero, Dallas; Gerald W. Gustafson, Indianapolis; C. Paul Hodgkinson, Detroit; Edwin S. Hoffman, Detroit; W. O. Johnson, Louisville; Arthur H. Klawans, Chicago; Harold W. Longyear, Detroit; J. H. Pratt, Rochester, Minn.; Ernest M. Solomon, Highland Park, Ill.; Howard J. Tatum, New Orleans; Howard C. Taylor, Jr., New York; Steward Taylor, Denver; and Clifford J. Vogt, Cleveland.

Drs. T. R. Hannon, chairman, and Herman R. Gardner, co-chairman, are making local arrangements.

PERSONALS

Dr. Minnie L. Maffett, Dallas, was recently elected one of the two vice-presidents of the American Medical Women's Association at the group's annual meeting in New York.

Dr. Arthur Grollman, Dallas, attended a July symposium on hypertension in London, England, and visited various laboratories in Europe.

At its annual meeting recently in New Orleans, the Association of Bone and Joint Surgeons elected *Dr. Louis W. Breck,* El Paso, second vice-president.

Dr. William K. Strother, Jr., Dallas, was one of the discussants by invitation at the annual meeting of the American Association of Obstetricians, Gynecologists, and Abdominal Surgeons in Hot Springs, Va., September 10-12.

At the annual meeting of the American Roentgen Ray Society in Cincinnati September 29-October 2, *Dr. Charles L. Martin,* Dallas, turned over the presidency to Dr. Harry M. Weber, Rochester, Minn.

Announcement was made September 15 of the appointment of H. Jack Roe, Kerrville, as executive director of the National Muscular Dystrophy Research Foundation by the president, *Dr. A. L. Delaney,* Liberty.

Dr. Bruce McMillan, Overton, in August was appointed to serve as a member of the board of trustees of Kilgore College.

Dr. W. E. York, Giddings, celebrated his eighty-third birthday August 9, with relatives from Brenham and Dallas in Giddings for the occasion.

A dinner and birthday cake with eighty candles marked the birthday of *Dr. J. E. Garner,* Turkey, on August 4.

On his seventy-fourth birthday, August 5, *Dr. E. P. Powell,* Centerville, was honored by the Centerville Chamber of Commerce in recognition of his service to the community and county.

Dr. H. L. Locker, his wife, and two sons, *Dr. S. Braswell Locker* and *Dr. Lewis H. Locker,* all of Brownwood, donated the $100,000 Main Hotel in Brownwood to the consolidated Howard Payne and Daniel Baker Colleges, it was announced July 3.

Dr. P. A. Baze, Mason, was honored August 27 for fifty years of medical service to Mason County. The arrangements committee included representatives from the Lions Club, the American Legion, the Chamber of Commerce, and the Junior Chamber of Commerce.

Andrew F. Moursund, Fredericksburg, died September 12 at San Angelo. He was the brother of *Dr. Walter H. Moursund,* Houston.

A. P. Cleveland, Austin, the father of *Dr. G. W. Cleveland,* Austin, was killed September 9 in an automobile-train crash.

Mrs. Irene Baldwin, mother of *Dr. Alvin Baldwin, Jr.,* Dallas, and *Dr. Russell E. Baldwin,* Tyler, died in July in a Memphis hospital.

Fall Out-of-State Meetings

Several out-of-state meetings scheduled for this fall may be of interest to the medical practitioner in Texas.

The fourth National Conference on Physicians and Schools of the American Medical Association was held September 30 and October 1 and 2 at Highland Park, Ill. Individaul discussions followed the theme of "Health Services for School Children."

The American College of Surgeons held its thirty-ninth annual clinical congress in Chicago from October 5 to 9. Subjects of the graduate courses conducted by leading surgeons were preoperative and postoperative care, surgery of the upper gastrointestinal tract, surgery of the small and large bowel, cardiovascular surgery, obstetrics and gynecology, and trauma to the abdomen.

The International Medical Assembly of the Interstate Postgraduate Medical Association of North America will be held November 2-5 in Chicago. Dr. Michael E. DeBakey, chairman of the Department of Surgery, Baylor University College of Medicine, Houston, is to be one of the scientific speakers. Details about the program and registration may be obtained from the general chairman, Dr. Walter C. Bornemeier, 55 East Washington Street, Chicago 2.

The Association of Military Surgeons has scheduled its sixtieth annual meeting for November 9-11 in Washington, D. C. This meeting will be devoted to subjects having direct military application, and attendants will be brought up to date on the latest developments of military medicine. Medical reserve officers can earn reserve credit points by attending the sessions. The Department of Defense, Office of Public Information, Washington 25, can supply more information.

"Clinical Electrocardiography and Recent Advances in Cardiovascular Diseases," a postgraduate conference sponsored by the Colorado Heart Association, University of Colorado School of Medicine, Fitzsimons General Hospital, and Colorado Department of Public Health, will be held the week of November 9-14 in Denver. For the first three days, which will be devoted to clinical electrocardiography, there will be no registration fee. However, for the last two and a half days, which will be concerned with recent advances in cardiovascular diseases, a registration fee of $15 for out-of-state physicians will be charged. Further information can be obtained from Edwin M. Goyette, Col., M. C., Fitzsimons General Hospital, Denver.

The National Society for Crippled Children and Adults will hold its annual convention in Chicago November 12-14. One of the speakers will be Dr. Edward J. McCormick, Toledo, Ohio, President of the American Medical Association. The program will include general sessions, seminars, exhibits, demonstrations, workshops, panel discussions, and social functions. Further information may be obtained from the society's headquarters, 11 South La Salle Street, Chicago 3.

STATE BOARD OF HEALTH

Dr. J. B. Copeland, San Antonio, was elected to a two-year term as chairman of the State Board of Health September 14 at the board's quarterly meeting in Austin. Dr. George Turner, El Paso, became vice-chairman, and J. P. Burden, a San Angelo sanitary engineer, was reelected secretary.

Three newly appointed members attended the meeting. They were Dr. F. W. Yeager, Corpus Christi; Dr. Hampton C. Robinson, Houston; and A. C. Sloan, D.D.S., a Dallas dentist.

The other members of the board include Dr. O. B. Kiel, Wichita Falls; E. C. Baum, D. O., an Austin osteopath; and J. T. Stryker, a Woodville druggist.

Postgraduate Courses at Southwestern

Southwestern Medical School of the University of Texas has announced its schedule of postgraduate courses to be presented in Dallas during the academic year. November 9-11, the second part of a course on treatment of emergencies will be given. The course, designed for the general practitioner, will include such topics as rupture of liver, rupture of spleen, injuries to kidneys, management of burns, treatment of shock, perforations of hollow viscera, treatment of pulmonary edema and of pulmonary embolism, cardiac arrest, management of diabetic coma, treatment of mechanical obstruction of bowel, management of paralytic ileus, treatment of acute anemia, acute pancreatitis, management of acidosis in children, reactions to anesthetic agents, and laryngospasm during anesthesia.

Members of the American Academy of General Practice will be given twenty hours' postgraduate credit for the course, for which instructors will be full-time and clinical faculty members of Southwestern. Registration should be made in advance through the office of the dean. Registration fee is $5 and tuition is $25.

Other courses on the agenda are on obstetrics and gynecology, December 7-9; systemic mycoses, January 18-20; orthopedic surgery, February 15-16; and surgery, April 19-21. Additional courses will be offered throughout the year one night a week for six to ten weeks at Dallas and other places where a sufficient number of physicians can be assembled.

Details may be obtained from Dr. John S. Chapman, Assistant Dean for Graduate and Postgraduate Education, 2211 Oak Lawn Avenue, Dallas.

Already completed is a course in psychosomatic medicine and everyday psychiatry, held October 5-7, sponsored jointly by Southwestern Medical School, the Postgraduate School of Medicine of the University of Texas, the Dallas Southern Clinical Society, the Texas Medical Association, and the Texas Academy of General Practice.

Honor guest was Dr. Stewart Wolf, Oklahoma City, professor of medicine, University of Oklahoma School of Medicine. He discussed "Physiologic Changes in Response to Emotional Stimuli and Pharmacology of Placebos." Other program participants were faculty members of Southwestern Medical School.

Conference on Residency Training in Pediatrics

A conference on residency training in pediatrics will be held at the University of Texas Medical Branch, Galveston, November 13-14. Sponsored by the Committee on Medical Education of the American Academy of Pediatrics, the program is divided into clinical sessions and seminars the first day and round-table discussions on residency training programs on the second day. Drs. Ralph V. Platou and Russell Blattner will represent the fifth region of the academy, and the Medical Branch's Department of Pediatrics, with Dr. Arild E. Hansen as chairman, will be host to the session.

An invitation is extended to any interested physicians.

Texas Neuropsychiatric Association

The Texas Neuropsychiatric Association has planned to alternate meetings annually with the Mexican Neuropsychiatric Society, with the meetings to be held one year in Mexico and the next in Texas. The fall meeting has been scheduled for November 6-7 in Mexico City.

A copy of the reservation, which should be made directly with the Hotel Bamer in Mexico City, should be sent to the secretary of the Texas group, Dr. John L. Otto, John Sealy Hospital, Galveston, so that discussants for the scientific program can be tentatively assigned.

FALL CLINICAL CONFERENCE

The North Texas-Southern Oklahoma Fall Clinical Conference was held September 16 in Wichita Falls with Drs. Jack S. Guyton, Samuel P. Asper, Jr., and Alan Churchill Woods, Jr., all from the faculty of Johns Hopkins University School of Medicine, Baltimore, and Dr. J. T. Grayhack, of the Johns Hopkins Hospital staff, as guest speakers.

Dr. Guyton's subject was "Treatment of Squint"; Dr. Asper spoke on "Some Considerations of Clinical Use of ACTH and Cortisone" and "Newer Aspects in Treatment of Thyroid Diseases"; Dr. Woods discussed "Surgical Treatment of Complications of Cirrhosis" and "Surgical Treatment of Cancer of Thyroid"; Dr. Grayhack's address was "Diagnosis and Treatment of Operable and Inoperable Prostatic Cancer." Round-table question and answer periods were a part of the program.

Dr. George J. Seibold and Dr. James Lee presided. Conference chairman was Dr. Charles H. Wilson.

SAN ANTONIO TUMOR SEMINAR

Dr. Frank Foote, pathologist at the Memorial Hospital in New York City, will be guest speaker and conductor of the annual San Antonio Tumor Seminar November 7 at the Brooke Army Hospital in San Antonio. It will be held in conjunction with the activities of the south central regional meeting of the College of American Pathologists. Under the sponsorship of the San Antonio Society of Pathologists, the seminar is open without fee to all pathologists.

Requests for slides may be sent to Col. Carl J. Lind, Department of Pathology, Brooke Army Hospital, Fort Sam Houston.

LIBRARY SECTION

HISTORY OF MEDICINE IN TEXAS

The arrival of Cabeza de Vaca, the first white doctor to set foot on Texas soil and practice the healing art, dates back to the year 1528. With this romantic beginning, Texas medicine has marched forward hand in hand with the progress of the state. Six flags have flown over this great state and medicine men have had an integral part in the developments. Dr. Anson Jones, the last President of Texas, proved a puzzle that kept foreign ministers and diplomats on two continents guessing. The story of the Alamo or the story of the entire state cannot be told unless it includes the history of medicine.

The following items with brief descriptions have been selected to show the real history of Texas medicine and are available for loan from the Library:

Anson Jones, The Last President of Texas, by Herbert Gambrell, Garden City, N. Y., Doubleday & Company, Inc., 1948. An adventurer in spite of himself; a puzzle to foreign ministers and diplomats on the two continents.

Biographical Sketch of Ashbel Smith, prepared at the request of the Ex-Presidents Association, by Mrs. S. C. Red, 1929. The man everyone knew.

The Medical Story of Early Texas 1528-1853, by Dr. Pat Ireland Nixon, Lancaster, Pa., Lancaster Press, 1946. Medicine and the men who made it from the early period to the formation of the Texas Medical Association.

A History of the Texas Medical Association 1853-1953, by Dr. Pat Ireland Nixon, Austin, University of Texas Press, 1953. A record from the formation of the Association to the present, which must be read to appreciate all the achievements of the Association.

The Medicine Man in Texas, by Mrs. S. C. Red, Houston, Standard Printing and Lithographing Company, 1930. Stories of the important men in medicine and state affairs from the early period through the Civil War and up to 1931.

A Century of Medicine in San Antonio, by Dr. Pat Ireland Nixon, Lancaster, Pa., Lancaster Press, 1936. The story of medicine in Bexar County for 100 years.

History of the Waco Medical Association, by Dr. William O. Wilks, privately printed in 1931. On April 30, 1866, ten doctors realizing the need for an association formed this society antedating the Texas Medical Association by three years.

A Doctor Looks at Life, by Dr. Witten Booth Russ, San Antonio, Naylor Company, 1952. A philosophical work by an outstanding surgeon and an ardent politician, whose writings have the religious and philosophical flavor that make up a part of the author's daily life. The youngest president the Texas Medical Association has had, Dr. Russ was in the forefront when the medical profession was urging the passage of adequate laws to protect health and promote sanitation early in the century. The cause of better health long has been his own personal fight.

Doctors and Doctors, Wise and Otherwise, by Dr. Charles M. Rosser, Dallas, Mathis Van Nort, 1941. A work with humor and philosophy; an authoritative first hand review of doctors on the firing line for half a century.

Pills and Proverbs, by Dr. Charles H. McCollum, Boston, Meador Publishing Company, 1941. An autobiography of a country doctor and his rise to be a great surgeon and important citizen in a modern city.

More Than Armies, by Booth Mooney, Dallas, Mathis Van Nort, 1948. The story of Dr. Edward H. Cary, an outstanding physician who has served as President of the American Medical Association, Texas Medical Association, and the Southern Medical Association. A dean for twenty-seven years, he is known as a courageous, gentle, friendly, and affectionate human being.

Fermentation, Infection and Immunity, by Dr. J. W. McLaughlin, Austin, Eugene Von Boeckmann, 1892. A theory new at the time of publication of processes which unified their primary causation and placed the explanation of their phenomena in chemistry, biology, and the dynamics of molecular physics. This volume is made up of articles previously published in *Daniel's Texas Medical Journal* and the *Texas Sanitarian.*

Yellow Fever in Galveston, Republic of Texas, 1839, Austin, University of Texas Press, 1951. An account of the great epidemic by Dr. Ashbel Smith, ex-surgeon general of the Texian Army, together with a biographical sketch by Chauncey D. Leake and stories of the men who conquered yellow fever.

A Manual of Practice for the Diseases of Texas, by Dr. F. C. Wilkes. Published at Chapel Hill, Texas, by Wilkes and Stone, 1866. A book written to explain and extend the use of a system of medicine recommended by the author.

Treatise on Eclectic Southern Practice of Medicine, by Dr. J. C. Massie, Philadelphia, Thomas, Cowperthwait and Company, 1854. A book written at the request of a group of the author's medical friends who felt the need of a book which would "present the various modifications which diseases assume in Texas." As stated in the preface, Dr. Massie's desire was to give his fellow physicians a textbook they would find

useful. In 1854 many doctors wanted a variety of remedies and Dr. Massie obliged with many pages of prescriptions.

Medical Consultation Book, by Dr. G. P. Hachenberger, Austin, Eugene Von Boeckmann, 1893. A pharmacologic and clinical book of reference containing the therapeutics of a full list of the official and nonofficial articles of the Materia Medica of the time with a consideration of the action of medicine, including an extensive collection of favorite prescriptions from the most reliable authorities of the medical profession; classified to be of ready access for authenticated treatment of each disease in its different stages and complications.

Additional Library Funds

The importance of endowment funds to the growth of the Texas Medical Association Memorial Library has been mentioned previously in recent months.* Although the Texas Medical Association supports from current income the operating expenses of the Library, interest on endowment funds may be expected eventually to mean the difference between the purchase of material which will permit the Library to provide outstanding service and the necessity of keeping expenditures to the bare minimum consistent with any sort of lending procedure.

Endowment funds which afford spendable income from the outset are valuable. However, the Library, looking to future needs, will derive much benefit from other contributions which are designed to begin bringing in spendable revenue some years after the initial gift. Two of the latter type of gift, interest from which will become available soon, are as follows:

Dr. D. H. Hudgins Memorial $740.00
Dr. Sterling E. Russ Memorial $740.00

BOOKS RECEIVED IN SEPTEMBER

Dill, L. B.: *The Obstetrical Forceps,* Springfield, Ill., Charles C. Thomas, 1953.

Grant, J. C.: *An Atlas of Anatomy,* ed. 3, Baltimore, Williams and Wilkins, 1953.

Rusk, Howard A., and Taylor, Eugene J.: *Living With a Disability,* Garden City, N. Y., Blakiston Company, 1953.

CONTRIBUTIONS TO LIBRARY

Grateful acknowledgment is made by the Texas Medical Association Memorial Library of a recent gift from Dr. Earl L. Yeakel, Jr., Austin, of 86 journals and 78 reprints.

MOTION PICTURE FILM LIBRARY

Be Your Age

16 mm., sound, 12 minutes. (Courtesy of Metropolitan Life Insurance Company, New York.)

This film is the story of a middle-aged, overweight man's recovery from a heart attack, and his adjustment to his handicapped heart. Emphasizing that heart disease is not necessarily fatal, it will be helpful to physicians in presenting programs to nonprofessional groups such as civic clubs.

Losing to Win

16 mm., sound, color, 11 minutes. (Courtesy of Metropolitan Life Insurance Company, New York.)

This film tells in an entertaining way the story of Mr. and Mrs. Chubby and their struggles to reach normal weight. In animated cartoon form it brings out the fact that overweight is dangerous and affects life expectancy, as well as personal well-being. This is an excellent motion picture for physicians presenting programs on this subject to any lay group. Statistics and program materials to aid in the presentation are available from the Library.

The Surgical Treatment for Coarctation of the Aorta

16 mm., silent, color. (Courtesy of Dr. Philip Thorek, Chicago.)

Surgical repair in which the coarctate area is severed and the proximal end of the aorta ligated is shown. An end to end anastomosis between the proximal end of the subclavian artery and the distal end of the aorta complete the operation. Roentgenograms, diagrams, and postoperative views amplify the film.

Feeding the Infant During the First Year

16 mm., sound, 25 minutes. (Courtesy of Mead Johnson, Evansville, Ind.)

In this film Dr. Alan Brown presents the problem of breast feeding of infants versus artificial feeding. This is an excellent motion picture for general practitioners.

Operative Procedures for Post-Poliomyelitis Paralysis

16 mm., sound, color, 50 minutes. (Courtesy of National Foundation for Infantile Paralysis, New York.)

Produced in the Orthopedic Division of Surgery, Duke University School of Medicine, this motion picture presents four operative procedures: the technique of epiphyseal arrest; open technique lengthening for contracture of the tendon achillis; technique for triple arthrodesis; and technique for knee fusion. This is an excellent film for orthopedists and general surgeons.

BOOK NOTICES

[1]Biochemistry of Disease

M. BODANSKY, M. D., *and* O. BODANSKY, M. D., Ph. D., *Professor of Biochemistry, Sloan-Kettering Division, Cornell University Medical College; Attending Clinical Biochemist and Chief, Clinical Biochemistry, Memorial Center for Cancer and Allied Diseases, New York. Second edition. 1,208 pages. $12. New York, The Macmillan Company, 1952.*

The first edition was written in 1939. Since then (1941) Meyer Bodansky has died. His brother, Oscar Bodansky, has almost completely rewritten the book, expanding the contents considerably, and brought it up to date. The original plan of organization, arrangement of the material according to clinical entities, has been maintained. Each chapter is headed by a complete table of contents, and at the end of each is an extensive bibliography, including many recent references.

The textbook is recommended as a reference for medical students, a basic science reference for residents in any field of medicine, and a library reference for physicians. Clinical pathologists will find it useful.

There are weak points in the book but considering its range, the author has handled the material exceptionally well.

[2]Visual Anatomy: Thorax and Abdomen

SYDNEY M. FRIEDMAN, M. D., Ph. D., *Professor of Anatomy, University of British Columbia, Vancouver; Formerly, Associate Professor of Anatomy, McGill University, Montreal. 203 pages. $10.50. Springfield, Ill., Charles C Thomas, 1952.*

This is the second volume of a series designed to present briefly and forcefully anatomy which the author feels is

*Library Endowment Funds, Texas State J. Med. 49:164 (March) 1953.

[1]*Harbert Davenport, Jr., M. D., Jacksonville.*
[2]*W. D. Seybold, M. D., Houston.*

"essential for the practice of medicine. The books are designed as a review for the student, undergraduate or postgraduate, who has already completed a dissection."

The presentation is primarily visual with the written word supplementary. There are ninety-one line and halftone drawings which have been executed by the author. Each illustration which is semidiagramatic in concept occupies the right half-page, while the corresponding description occupies the left half-page. The drawings are clear, simple, and concise, and the work combines in an effective way the features of a descriptive text with those of an atlas of anatomy.

As a means of a general review in the anatomy of the thorax and abdomen or as a companion volume to a standard textbook, this work can be recommended. It is not a book that will meet the needs of the surgeon or of the postgraduate student in surgery.

[3]Practical Clinical Chemistry

ALMA HILLER, Ph. D., *Associate Attending Biochemist in Charge of Clinical Chemistry, The Presbyterian Hospital of the City of Chicago; Associate Professor of Biological Chemistry, University of Illinois College of Medicine, Chicago. 266 pages. $6.50. Springfield, Ill., Charles C Thomas, 1953.*

This guide for chemical technicians is divided into two parts, the first consisting of 211 pages with the index and the second of 48 pages of the "Outlines" only of the twenty-four methods. These outlines, usually covering a page or so, can be removed from the book, and put into suitable holders, and hung in different parts of the laboratory for constant use. In a large laboratory this is a desirable feature. There are 13 tables included.

The technician can master the chapter on a given method in the first part and uses the outlines in the actual determinations. General laboratory procedures, detailed calculations, the preparation of volumetric solutions, the use of spectrophotometers, that is, the Coleman, Jr. spectrophotometer, are given, together with the normal range of the different values determined in the blood and urine.

Results of laboratory reports at present leave much to be desired. This excellent guide should be in every clinical chemistry laboratory; it also can serve as a reference textbook for courses in clinical chemistry. At present more and different types of chemical determinations, such as the determinations of vitamins, hormones, sterols, and protein-bound iodine, are becoming of increasing clinical importance. Also, methods for the detection of poisons, such as mercury, lead, arsenic, phosphorus, alcohol, and barbiturates, should be included. It is hoped that the author will issue a companion volume covering these subjects.

[4]Symptoms and Signs in Clinical Medicine

E. NOBLE CHAMBERLAIN, M. D., M. Sc., F.R.C.P., *Senior Lecturer in Medicine, University of Liverpool; Senior Physician, Royal Southern Hospital, Liverpool; Physician in Charge, Medical Unit, Sefton General Hospital, Liverpool. Fifth edition. 480 pages. $8. Baltimore, Williams and Wilkins Company, 1952.*

As the title implies, accounts of the common symptoms and physical signs of disease have been given, but the author, believing that these are often wrongly divorced from diagnosis, has attempted to take the student a stage further to consider symptoms and signs as forming a clinical picture of a pathologic process.

Dr. Chamberlain's book deals with the various systems of the human body, each described briefly yet understandably.

I am particularly impressed with the first and second chapters which deal with the routine of interrogation and examination and external characteristics of disease. The book is written in a simple, understandable, and thorough style so that the interpretation of the signs and their correlation with the symptoms described in the succeeding chapters of the various systems is the basis for diagnosis.

The book is particularly valuable to the physician who is first introduced to the practice of medicine and certainly is a refresher and guide to the experienced physician who has neglected some of the important facts in making a final and correct diagnosis. It should be in the library of every physician, be he a general practitioner or a specialist, and should be referred to frequently, even by those who are most experienced.

[5]A Manual of Clinical Allergy

JOHN M. SHELDON, M. D., *Professor of Internal Medicine, University of Michigan Medical School, Ann Arbor; Assistant to the Chairman of the Department of Postgraduate Medicine; Physician in Charge of University of Michigan Allergy Clinics;* ROBERT G. LOVELL, M. D., *Instructor in Internal Medicine, University of Michigan Medical School; and* KENNETH P. MATHEWS, M. D., *Assistant Professor of Internal Medicine, University of Michigan Medical School. 413 pages. $8.50. Philadelphia, W. B. Saunders, 1953.*

The authors present a practical concise volume intended for the physician devoting only part of his time to the study of allergy. The book contains apparently well prepared sections on preparation of extracts, pollen identification, patch testing, office equipment, aerosol therapy, contact allergy, and food allergy. Lengthy theoretical discussions usually found in other texts on the subjects are avoided, although sufficient emphasis is given to theoretical consideration as a basis for treatment.

In the reviewer's opinion this is an excellent basic textbook in the field, yet it contains specific direct instruction for workup, management, and treatment of allergic disorders of special value to the practitioner.

[6]Battle Casualties

GILBERT W. BEEBE, Ph. D., *Division of Medical Sciences, National Research Council, Washington, D. C., Formerly Captain, Medical Administrative Corps, A.U.S.; and* MICHAEL E. DEBAKEY, M. D., *Professor of Surgery, Baylor University College of Medicine, Houston, Formerly Colonel, Medical Corps, A.U.S. 277 pages. $10.50. Springfield, Ill., Charles C Thomas, 1952.*

The authors have interpreted much unplanned and partly unreliable observations in 734,000 World War II battle casualties, despite the fact that most statisticians would despair at the unpreciseness of field records.

Killing power increased, but the World War II enemy was only 60 per cent as effective considering battle deaths per thousand per year as was the World War I enemy, the comparative figures being 10.1 per thousand for World War II and 16.4 per thousand for World War I. The number of those who died of wounds was significantly changed, the figures per thousand per year being 1.1 for World War II as against 4.4 for World War I. Total deaths numbered 12.9 per thousand per year for World War II as against 34.3 for World War I.

Psychiatric and psychosomatic breakdowns tended to correlate with combat casualties, as did such environmental factors as cold. Wounded in action and killed in action ratios for ground troops have varied little in more than two

[3]*H. H. Beard, Ph. D., Fort Worth.*
[4]*A. W. Brazda, M. D., Ranger.*
[5]*A. I. Exline, M. D., Austin.*
[6]*Lee D. Cady, M. D., Houston.*

hundred years, the ratio being about 4.1 wounded to 1 killed; these ratios differed between the various arms or services, in assaults and retreats, and in "Military Occupation Specialties," however.

There is a significant variation in deaths from wounds between wars and theaters of World War II: In the Mexican War 14.9 per cent of the wounded died; in World War I (excluding gas) 8.1 per cent; and in World War II 4.5 per cent (recent Korean reports give a figure of 2.5 per cent). A "surgical paradox" is the fact that the farther forward surgery was pushed (the earlier the wounded got surgery), the higher became the death from wounding rate; another is that body armor reduced the chest wound incidence but increased the incidence of other injuries.

The book contains splendid considerations of logistics as regards personnel, hospitalization, and evacuation, together with Dr. Edward D. Churchill's "thirteen propositions" on evacuation and distribution of battle casualties.

ORGANIZATION SECTION

TEXAS MEDICAL ASSOCIATION

Miss Keith Retires from Staff

After more than thirty years as a member of the central office staff of the Texas Medical Association, Miss Anna Keith has retired from her position as head of the business and membership department and advertising manager of the TEXAS STATE JOURNAL OF MEDICINE. Miss Keith came to the Association in Fort Worth in June, 1923, when a staff of four, including the porter, was able to take care of the needs of the approximately 3,700 members. She watched the organization grow to a membership of 6,500 with a greatly expanded program of activity assisted by an employed staff of twenty.

MISS ANNA KEITH

Recognition of Miss Keith's loyalty and service has been given twice in recent months. The Board of Trustees presented her with a wrist watch in observance of her thirtieth anniversary with the Association, and Dr. and Mrs. Sam E. Thompson of Kerrville gave a silver goblet in her name for use in the Sam Thompson Room of the headquarters building.

TELEPHONE POSTGRADUATE BROADCASTS

The final program in the series of telephone postgraduate broadcasts sponsored by the Texas Medical Association will be presented November 10 at 8 p. m. The topic of discussion will be the "Use of Antibiotics." Dr. James E. Kreisle, Austin, will be moderator; members of the panel and their topics follow: Dr. W. V. Leary, Houston, "Use of Antibiotics in Respiratory Tract Infections, Including Tuberculosis"; Dr. Edgar J. Poth, Galveston, "Use of Antibiotics in Surgical Conditions with Special Reference to Intestinal Antisepsis and Complications of Oral Administration"; Dr. Andres Goth, Dallas, "Fundamentals of Antibiotic Action"; and Dr. Robert L. Moore, Dallas, "Use of Antibiotics in Pediatric Practice."

Thirty-four county medical societies participated in the October 13 discussion of "Obstetric Emergencies." Moderator was Dr. William F. Mengert, Dallas. The following physicians served on the panel: Dr. Van D. Goodall, Clifton, whose subject was "Obstetric Emergencies as Seen in General Practice"; Dr. W. Grady Reddick, Dallas, "Obstetric Emergencies Relating to Heart, Diabetes and Anemia"; Dr. S. Foster Moore, San Antonio, "Surgical Emergencies Attending Reproduction"; and Dr. Robert A. Johnston, Houston, "Obstetric Hemorrhage."

EXECUTIVE COUNCIL CONVENES

The semiannual meeting of the Executive Council of the Texas Medical Association was held at the headquarters building in Austin on September 13 with Dr. George Turner, El Paso, President, presiding.

Following Dr. Turner's introductory address, in which he reviewed the present status of governmental care of veterans with nonservice-connected disabilities and the opposition of the medical profession and of the veterans themselves to such comprehensive care, he presented a plaque to Dr. Clarence M. Cash, San Benito, elected General Practitioner of the Year at the 1953 annual session.

The council then unanimously adopted a resolution condemning the action of a hospital administration which had canvassed the members of the local county medical society asking for sizable cash donations to assist in a building program and threatening to make such donations the basis for hospital staff privileges.

Reports from the various boards, councils, and committees of the Association then were heard. Among the reports were the following items:

Board of Trustees.—Anticipated revenues for 1954 are $330,000 with anticipated expenditures less than $300,000. The loan on the Association's building, completed in September, 1952, for $415,000, with an additional payment authorized for October 1 of $30,000 plus the regular monthly payment was to be reduced in mid-October to $267,840. Prizes of $250 for state winners in the 1954 essay contest for high school students sponsored nationally by the Association of American Physicians and Surgeons have been approved.

Council on Medical Defense.—Attention was called to a booklet, "How to Avoid Malpractice Suits," distributed to members of the Association.

Council on Medical Jurisprudence.—A mimeographed report on legislation enacted by the Fifty-Third Texas Legislature or pending in Congress was to be distributed to members of the Executive Council and officers of the Woman's Auxiliary. Copies of this report may be obtained upon request from the central office.

Council on Scientific Work.—Plans for the 1954 annual session to be held in San Antonio, May 3, 4, and 5 were outlined. Guest speakers already obtained were announced as Drs. Simeon T. Cantril, Seattle, radiologist; George T. Harrell, Jr., Winston-Salem, internist; Harold D. Palmer, Denver, pediatric pathologist; Robert L. Sanders, Memphis, surgeon; and Edward S. Taylor, Denver, obstetrician and gynecologist.

Council on Medical Education and Hospitals.—Attention was called to the shortage of interns, residents, and nurses, and the cooperation of members of the Association in making the educational opportunities for such personnel particularly attractive in Texas was requested. Attention was called to the four vacancies existing on the advisory committee for Hill-Burton Act activities in the State Health Department and careful selection by the Governor of personnel to fill these vacancies was recommended. Announcement was made that thirty-two societies had signed up for the postgraduate telephone broadcasts to be presented October 13 and November 10 under sponsorship of the Council on Medical Education and Hospitals and that more than seventy societies have local chairmen for promotion of the American Medical Education Foundation.

Committee on Public Relations.—Cooperating with other committees of the Association, the Committee on Public Relations has continued to stress mediation committees, around-the-clock emergency medical service, training of medical secretaries and clerical assistants, and education of county society presidents and secretaries and public relations chairmen. The committee also assisted in the first medical students day held at Southwestern Medical School of the University of Texas in Dallas, is cooperating in a survey to determine the adequacy of doctor distribution, is helping the Woman's Auxiliary produce a handbook for its officers and committee chairmen, and is assisting in the publication of special newspaper sections in a score of cities in observance of the centennial of a statewide medical organization in Texas.

Committee on Rural Health.—The establishment of local health committees, with the blessing of the Texas Medical Association and assistance from the Extension Service of Texas Agricultural and Mechanical College, was recommended to help solve health problems in rural areas.

Committee on Doctor Distribution.—A survey of doctor distribution as viewed by the medical profession has been completed, and a pilot survey, to be extended throughout the state later, is now being carried on to determine whether community leaders believe a shortage of doctors exists in their area and whether in reality such a shortage prevails.

Executive Secretary.—Membership in the Association as of September 10 was announced as totaling 6,550, an increase of more than one hundred over the same figure last year. January 24 was set for the next meeting of the Executive Council.

Also called to the attention of council members was a report of the Committee for the Study of Relations Between Osteopathy and Medicine presented at the 1953 session of the American Medical Association and referred to the states for study prior to action next year. Copies of this report may be obtained from the central office.

Mrs. E. W. Coyle, San Antonio, President of the Woman's Auxiliary to the Texas Medical Association, spoke briefly to the council, offering again the full cooperation of the Auxiliary in furthering the objectives of the medical profession.

General Practitioner of Texas

A bronze wall plaque commemorating his selection as the General Practitioner of Texas for 1953 was presented September 13 to Dr. Clarence McClellan Cash of San Benito.

The presentation was made by Dr. George Turner, El Paso, President of the Texas Medical Association, during a meeting in Austin of the Executive Council. Dr. Cash, who was chosen in April by the Association's House of Delegates to receive the award, is the fifth Texas physician to be so honored.

Born in White County, Tenn., on April 19, 1864, Dr. Cash came to Texas at the age of 12 years. Two years later, he was enrolled in Southern Illinois Normal University, near Carbondale. When he was 16 years old, he returned to Texas and began earning his living by working as a farm hand.

Dr. Cash began teaching school at Kosse in Limestone County in 1884. Because of ill health, he moved to West Texas in 1890, settling at Knickerbocker. Simultaneously from 1893 through 1896, he taught school at Knickerbocker and served a medical preceptorship under Dr. Boyd Cornick. After studying medicine at old Fort Worth Medical College in Fort Worth, he first practiced medicine in 1898 at Guion. He conducted his practice on horseback from saddlebags given him by a retiring physician. Dr. Cash later succeeded in founding the town of Tuscola, which is near Guion.

DR. C. M. CASH

In 1906 he and Dr. L. W. Hollis became partners in the Hollis Sanitarium at Abilene, the first hospital to be established between Fort Worth and El Paso. While in Abilene, he served as city and county health officer and helped draft the first milk ordinance for Abilene.

Dr. Cash moved to San Benito in 1914. One year later, he began occupying the same offices which he uses today in his active medical practice. He has served as chief of the obstetric service of the Valley Baptist Hospital in Harlingen and was chief of staff there in 1928. He held a similar position at the Dolly Vinsant Memorial Hospital in San Benito in 1950. Dr. Cash was instrumental in organizing the Cameron County Health Unit.

Throughout his medical career, Dr. Cash has taken numer-

ous postgraduate and refresher courses. One of the most recent courses he took was one in obstetrics, for which he enrolled on his eighty-fifth birthday in 1949. He has taken a refresher course nearly every year.

Dr. Cash has been a member continuously since 1905 of the American Medical Association and the Texas Medical Association, first through the Taylor and later through the Cameron-Willacy Counties Medical Societies. He has served as secretary, vice-president, a member and chairman of the board of censors, and twice as president of the latter group, in addition to serving eight years as a delegate to the State Association.

Long active in civic work, Dr. Cash was mayor in 1916 and again in 1928 of San Benito. From 1922 to 1928 he was a city commissioner. He is a charter member and a past president of the San Benito Rotary Club, and has been named to honorary membership in the Kiwanis, Lions, and 20-30 Clubs of San Benito. In 1940 the citizens of the community presented him with a plaque in recognition of his many years of service to them. The citizens of San Benito in September, 1952, held a "Dr. Cash Day." At that time a new elementary school was dedicated and named in his honor. Also in 1952, the Woman's Auxiliary to the Cameron-Willacy Counties Medical Society presented him with a plaque signifying his fifty-four years of medical practice.

For more than fifty years, Dr. Cash has been active in Masonic work. In 1894 he was made a Master Mason in the Winters Lodge at Winters. He was master of the Tuscola Lodge in 1903, and master of the San Benito Lodge in 1916. He has held a series of high offices in Masonry, culminating in his being made a thirty-third degree Scottish Rite Mason at Washington, D. C., in 1949.

In 1884 Dr. Cash married the former Miss Theodosia Stone of Blue Ridge. She was drowned in the 1919 hurricane in Corpus Christi. He married the former Mrs. Mona Reed in 1925. Dr. Cash has four children, two daughters, Mrs. Paul Cottrell, San Benito, and Mrs. Ruth Edwards, Fort Sill, Okla., and two sons, Clarence M. Cash, Jr., D.D.S., Beaumont, and Dr. Auda V. Cash, Abilene, also a member of the Texas Medical Association.

NEW BUSINESS MANAGER APPOINTED

The new business manager of the Texas Medical Association is E. Maxwell Jones, who had been office manager of the Texas State Board of Vocational Nurse Examiners since the establishment of the board in September, 1951, until his recent resignation. Mr. Jones, who has a master of business administration degree from the University of Texas, was employed as assistant state auditor from 1949 to 1951 after moving to Austin from Houston, where he was associated with J. A. Phillips, Sheffield and Company, certified public accountants. His wife, Mary, is executive secretary of Travis County Medical Society.

AMERICAN MEDICAL ASSOCIATION

VETERANS MEDICAL CARE TO BE DISCUSSED

A regional meeting has been scheduled for November 6 in Dallas to follow through on the discussions begun at the American Medical Association's Conference on Veterans Medical Care, held in Chicago September 1, regarding veterans with nonservice-connected disabilities. Representatives from Texas, Louisiana, Oklahoma, Arkansas, and Mississippi are to attend the meeting to develop a working liaison with state committees dealing with veterans' problems, to discuss methods for carrying out the instructions of the AMA House of Delegates regarding veterans' medical care, to increase the number of physicians who have a working knowledge of the policy and the facts relating to it, and to learn the situation in each state relative to nonservice-connected disabilities.

Those members of the Texas Medical Association who have been invited to attend the one-day informal meeting are the officers, the Council on Medical Jurisprudence, the Council on Medical Economics, the Board of Councilors, the Board of Trustees, the delegates to the AMA, and county society presidents and secretaries.

Other regional meetings are to be held in Atlanta, Ga., November 8; in New York, November 13; and in Washington, D. C., November 15.

COUNTY SOCIETIES

Bexar County Society

September 24, 1953

Approximately four hundred members of the Bexar County Medical Society, its auxiliary, and guests participated in a garden reception at the society's library in San Antonio the evening of September 24. The occasion was the one hundredth anniversary of the founding of the society.

Mr. Tom Keys, librarian of the Mayo Clinic, Rochester, Minn., spoke on the history of medicine, basing his talk on slides of the stained glass window at Mayo Foundation House, Rochester, which depicts major steps in medical education, medical practice, and medical research from 500 B. C. to 1900 A. D. Mr. Keys was introduced by P. I. Nixon, medical historian, who has been instrumental in the development of a large collection of rare medical books owned by the Bexar society and on display for the evening. Merton M. Minter, president-elect of the society, presented special guests, and E. W. Coyle, president, presided.

Those previously named and their wives, plus Mrs. John Parsons, president of the Woman's Auxiliary to the Bexar County Medical Society, and Dr. Parsons, were in the receiving line. Coffee and cake, the latter decorated with a gold caduceus and 100 candles, were served on the lawn.

Howard-Martin-Glasscock-Borden-Scurry-Kent-Dickens-Garza-King-Stonewall Counties Society

July 28, 1953

Donald McDonald, Roy T. Lester, and Travis Smith discussed the diagnostic and treatment methods of the polio center in the Hendrick Memorial Hospital, Abilene, at the July 28 meeting of the Howard-Martin-Glasscock-Borden-Scurry-Kent-Dickens-Garza-King-Stonewall Counties Medical Society in Big Spring. The three doctors are from Abilene.

The symposium followed a dinner which approximately twenty doctors and their wives attended. Floyd Mays, Big Spring, president of the local society, presided.

The group went on record as favoring annual chest roentgen-ray examinations for food handlers. The society decided to endorse a program in September in cooperation with the local press for the observance of the Texas Medical Association's one hundredth anniversary.

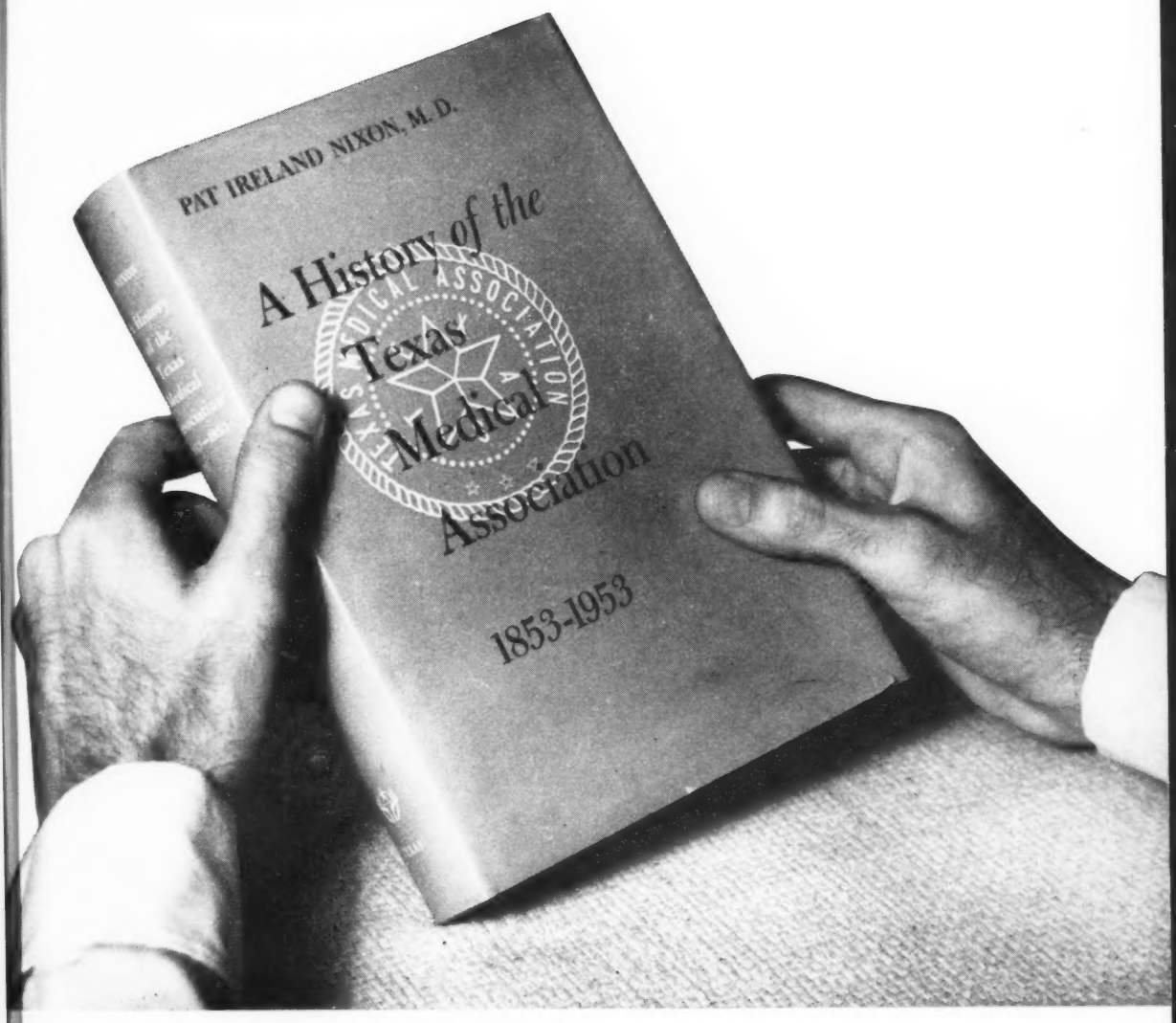
Kerr-Kendall-Gillespie-Bandera Counties Society

September 14, 1953

(Reported by Russell E. Guill, Secretary)

Resuscitation of Newborn—Benjamin B. Shaver, San Antonio.

Eighteen members of the Kerr-Kendall-Gillespie-Bandera Counties Medical Society heard the above discussion by Dr. Shaver at the society's regular monthly meeting September 14 in Kerrville.



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AUXILIARY SECTION

WHY AN AUXILIARY?

The purpose in having an organized Woman's Auxiliary to the Texas Medical Association may be said to be summed up in the last lines of the "Credo of a Doctor's Wife" by Mrs. Frank Haggard of San Antonio: "I promise to uphold the dignity of my husband's profession, and will help him render service to humanity in seeking to secure for him the free and unfettered practice of medicine."

Dr. George Turner, El Paso, President of the Texas Medical Association, recently had the following to say about the importance of the Auxiliary:

"To me, the Woman's Auxiliary to the Texas Medical Association is the greatest contributing force of the Association in accomplishing the many and complex problems confronting organized medicine in Texas today. You are, indeed, our help and support. Since the time your organization began, your accomplishments have been outstanding and amazing in helping to preserve our system of medical care. Unscrupulous planners have unceasingly sought to tear it down, but they failed to reckon with our Auxiliary force.

"In all fields of action to engender in the public mind a feeling of confidence, trust, and security in the supreme effort of the medical profession to adequately meet the needs of all people, at all times, your actions and performance stand in testimony of your worth. . . .

"The Auxiliary is the greatest undeveloped resource the medical association has, and public relations is your greatest field for accomplishment. Medicine has become an important force in the country, and we need your help in interpreting our aims and policies to the public. . . ."*

Dr. Turner's views closely parallel those expressed by Dr. John W. Cline, a past president of the American Medical Association. Dr. Cline, in a speech at the twenty-ninth annual meeting of the Woman's Auxiliary to the American Medical Association, cited the many values of an Auxiliary by emphasizing the projects carried on by it to achieve worth-while objectives. He said, for example, that the American Medical Association has earned the respect of many lay groups because of the fine citizenship represented by the medical profession and its Auxiliary. According to him, wives of physicians often have a more penetrating perception of values than do physicians.

"It is a tremendous asset for the medical profession to have a body, such as your organization, to do some straight moral thinking and to assist us in arriving at better conclusions when dealing with perplexing problems," Dr. Cline stated.

One Texas physician, Dr. B. E. Park, Dallas, has said, "Women are lifegivers, not consumers, and as all life is one, women by merely remaining true to their lifegiving genius are sure to remain the most effective workers in the designs of men's lives."

Address of Dr. George Turner, Texas State J. Med. 49:576-577 (July) 1953.

Officers of the Woman's Auxiliary to the Texas Medical Association: President, Mrs. E. W. Coyle, San Antonio; President-Elect, Mrs. Mark H. Latimer, Houston; First Vice-President (Organization), Mrs. John D. Gleckler, Denison; Second Vice-President (Physical Examinations), Mrs. Cecil O. Patterson, Dallas; Third Vice-President (Today's Health), Mrs. Ralph B. Payne, Amarillo; Fourth Vice-President (Program), Mrs. Guy Knolle, Houston; Recording Secretary, Mrs. Paul Brindley, Galveston; Corresponding Secretary, Mrs. Charles L. McGehee, San Antonio; Treasurer, Mrs. Oscar M. Marchman, Jr., Dallas; Publicity Secretary, Mrs. Allen H. Neighbors, Jr., Austin; Parliamentarian, Mrs. H. S. Renshaw, Fort Worth.

EXECUTIVE BOARD MEETING

The Executive Board of the Woman's Auxiliary to the Texas Medical Association met in the Sam Thompson Room of the Texas Medical Association Library and Headquarters Building, Austin, at 9 a. m. Thursday, September 10, 1953, with Mrs. E. W. Coyle, San Antonio, President, presiding.

Mrs. Haskell D. Hatfield, El Paso, gave the invocation. Members and guests were welcomed to Austin by Mrs. Charles Dildy, Austin, president of the Travis County Auxiliary; and Mrs. Paul Brindley, Galveston, responded on behalf of the Executive Board. The entire group stood and read in unison the Auxiliary Pledge.

A motion by Mrs. S. F. Harrington, Dallas, to dispense with reading the minutes of the last meeting, which were approved by a committee and published in the July JOURNAL, was carried.

Upon the motion of Mrs. Carlos R. Hamilton, Houston, the following committee, appointed to approve the minutes of this meeting, was confirmed: Mrs. John K. Glen, Houston; Mrs. H. S. Renshaw, Fort Worth; and Mrs. Charles L. McGehee, San Antonio.

Upon the motion of Mrs. O. M. Marchman, Jr., Dallas, the Board ratified the President's action in accepting the resignations of Mrs. W. F. Armstrong, Fort Worth, and Mrs. John C. Kuppinger, Harlingen, from the Nominating Committee and the appointment of Mrs. Ivan H. Readinger, Fort Worth, and Mrs. Troy Shafer, Harlingen, to fill the two vacancies.

In the absence of the Reference Committee, on motion of Mrs. H. Leslie Moore, Dallas, the following were appointed to serve for this meeting: Mrs. G. V. Brindley, Temple; Mrs. John H. Wootters, Houston; and Mrs. Robert F. Thompson, El Paso.

Mrs. Coyle spoke briefly of her wish to be of help to each member of the Board and each auxiliary. She stressed that she would be available upon request and particularly asked that the newly organized auxiliaries feel free to call upon her at any time.

The President-Elect, Mrs. Mark H. Latimer, Houston, expressed her appreciation to the Auxiliary for sending her to New York to her first meeting of the national organization. She pointed out that Texas is entitled to forty plus delegates to the meeting of the Auxiliary to the American Medical Association, and she urged that more Texas members register at the AMA meeting next summer in San Francisco.

Mrs. John D. Gleckler, Denison, First Vice-President and chairman of Organization, introduced the seven district Council Women who were present, each of whom gave a report: Mrs. Harold Lindley, Pecos; Mrs. E. K. Jones, Wellington; Mrs. June Yates, Corpus Christi; Mrs. Thomas J. Archer, Austin; Mrs. Jack Crow, Abilene; Mrs. James Atchison, Gainesville; and Mrs. D. C. Simmons, Kilgore.

Mrs. O. M. Marchman, Jr., Dallas, Treasurer, reported a balance of $11,796.43 on April 23; receipts of $294.00, and disbursements of $1,851.96, leaving a balance September 8 of $10,148.47.

The Corresponding Secretary, Mrs. Charles L. McGehee, San Antonio, read a letter from Mrs. George Turner, El Paso, President-Elect of the Auxiliary to the American Medical Association, expressing her appreciation to members of the Texas Auxiliary for the beautiful orchid given her in New York upon her election and for their love and cooperation in the Auxiliary program through the years. Mrs. McGehee reported messages from past presidents and others

who were unable to attend the meeting. Upon motion of Mrs. S. F. Harrington, Dallas, the Board voted to send a telegram to each absent past president.

Other officers present and reporting were Mrs. Cecil O. Patterson, Dallas, Second Vice-President and chairman of Physical Examinations; Mrs. Guy E. Knolle, Houston, Fourth Vice-President and chairman of Program; Mrs. H. S. Renshaw, Fort Worth, Parliamentarian; Mrs. A. H. Neighbors, Jr., Austin, Publicity Secretary; and Mrs. Paul Brindley, Galveston, Recording Secretary.

Mrs. Frank N. Haggard, San Antonio, Honorary Life President and chairman of the Special Advisory Committee, introduced each member of her committee present, all past presidents: Mrs. G. V. Brindley, Temple; Mrs. F. F. Kirby, Waco; Mrs. S. F. Harrington, Dallas; Mrs. P. R. Denman, Houston; Mrs. A. B. Pumphrey, Fort Worth; Mrs. George Turner, El Paso; Mrs. S. M. Hill, Dallas; and Mrs. Robert F. Thompson, El Paso.

The meeting was recessed briefly at 10:40 a. m. for coffee in the Reading Room, a courtesy of the Travis County Auxiliary, and to see an exhibit of early Auxiliary records, pictures of past presidents, and biographical data prepared by the Archives Committee, Mrs. F. F. Kirby, Waco, chairman, in cooperation with the staff of the central office.

The session was reconvened at 11:10 a. m. The President introduced each of the members of the permanent staff of the Texas Medical Association employed in the central office in Austin.

Dr. and Mrs. Sam Thompson, Kerrville, presented to the Texas Medical Association for use in the Medical Library, a sterling silver tray and a goblet. The tray was given in honor of Dr. Merton M. Minter of San Antonio for his outstanding work in the building of the Medical Library in Austin during his term as chairman of the Board of Trustees of the Texas Medical Association. The goblet was given in recognition of Miss Anna Keith's thirty years of faithful service to the Association. Because of Dr. Thompson's illness, neither he nor Mrs. Thompson was able to make the presentation. They were represented by Mrs. L. A. Feller, Fredericksburg, president of the Kerr-Kendall-Gillespie-Bandera Counties Auxiliary. The silver was accepted with expressions of deep appreciation to the Thompsons by Mrs. Coyle; Mr. N. C. Forrester, Executive Secretary of the Association; and Miss Keith.

The Executive Board voted, after a motion by Mrs. Ramsay H. Moore, Dallas, to send Dr. and Mrs. Thompson a telegram of appreciation, telling them of its pleasure in holding its first meeting in the Sam Thompson Room and its gratitude for their many courtesies in the past.

Mrs. Travis Smith, Abilene, chairman of History, recommended that each county president see that the history of her auxiliary is brought up to date this year; that awards be made in several new fields of endeavor; and that the state chairman in each field make recommendations to the judges for the awards.

Mrs. John K. Glen, Houston, chairman of Legislation, emphasized the necessity of keeping abreast of legislation pertaining to medicine. Upon the motion of Mrs. Glen, the Executive Board went on record as approving the Bricker amendment.

The business meeting was recessed from 12 noon until 1:30 p. m. for luncheon. Mrs. A. H. Neighbors, Jr., was chairman, assisted by Mrs. Charles Dildy, Mrs. S. W. Bohls, and Mrs. Charles Pelphrey, all of Austin.

Business was resumed promptly at 1:30 p. m. Mrs. Haskell D. Hatfield, El Paso, editor of the *News Letter,* explained the purpose, policy, and contribution of this organ. She recommended that $2,000 be appropriated for the cost of publication this year.

The chairman of Public Relations, Mrs. V. J. Simmon, Temple, presented a draft of the proposed Auxiliary Handbook, explaining that it had been prepared in cooperation with Syers, Pickle and Winn, the public relations counsel. She showed a dummy make-up and explained the purpose and cost, which should not exceed $1,234. Mrs. J. Peyton Barnes, Houston, moved, and the Board voted to publish the Handbook.

Other chairmen reporting were Mrs. Mal Rumph, Fort Worth, Civil Defense; Mrs. John C. Parsons, San Antonio, Convention; Mrs. G. V. Brindley, Temple, Memorial Services; Mrs. A. B. Pumphrey, Fort Worth, Mental Health; Mrs. James C. Terrell, Stephenville, Nurse Recruitment; Mrs. J. L. Jinkins, Galveston, American Medical Education Foundation Fund; Mrs. S. F. Harrington, Dallas, Library Fund; Mrs. John H. Wootters, Houston, Student Loan Fund; Mrs. J. Peyton Barnes, Houston, Research; Mrs. Ramsay H. Moore, Dallas, School of Instruction; and Mrs. R. T. Travis, Jacksonville, Yearbooks.

In the absence of Mrs. A. O. Severance, San Antonio, Essay; Mrs. J. Guy Jones, Dallas, Memorial Fund; Mrs. Edward C. Ferguson, Beaumont, Revisions; and Mrs. E. H. Marek, Yoakum, Bulletin, their reports were filed.

Twenty-four county presidents were present and presented their plans for the year.

The following recommendations presented by Mrs. H. Leslie Moore, Dallas, chairman of Finance, were adopted:

1. That the maximum of $1,240 be paid for 500 Handbooks.

2. That the Archives Committee be instructed to purchase a four-drawer filing cabinet, approximate price to be $140.

3. That the Auxiliary buy a ring-binder for each member of the Executive Board to be used from year to year to contain material pertinent to her office, these binders to be the property of the Auxiliary and passed on to successors in office.

4. That four recommended motion picture films be purchased from the Special Fund and kept in the Medical Library.

5. That the *News Letter* editor be allowed $2,000 maximum for the year.

The President read a suggestion from Mrs. O. W. Robinson, Paris, Past President, that the $5 check which she received from the Southern Medical Auxiliary for the outstanding Doctor's Day observance in Texas in 1951-1952 be given to the Jane Todd Crawford Memorial Fund of the Southern Auxiliary. This was moved by Mrs. H. Leslie Moore, Dallas, and carried.

Mrs. S. W. Bohls, Austin, chairman of Registration, reported that a total of 108 attended the meeting and luncheon: 10 officers, 22 committee chairmen, 14 committee members, 7 council women, 24 county presidents, 25 staff members, and 6 guests.

Mrs. Joseph H. McCracken, Jr., Dallas, presented the following resolution: "It is indeed a pleasure for the Executive Board of the Woman's Auxiliary to the Texas Medical Association to have this lovely building in which we are privileged to meet. We of the Resolutions Committee, in the name of our Board, wish to express our appreciation to Mrs. Coyle, Mrs. Dildy, and all the officers and members of the Travis County Medical Auxiliary, and to the staff of the Texas Medical Association. We say 'thank you' for a delightful time." Signed by Mrs. S. M. Hill, Dallas; Mrs. Carlos R. Hamilton, Houston; Mrs. Newton F. Walker, El Paso; and Mrs. McCracken.

The address of Mrs. George Turner, El Paso, President-Elect of the Auxiliary to the American Medical Association, was the highlight of the day's meeting. She ably developed her theme, "Know Your Auxiliary—Serve Your Commu-

nity," into an inspiring piece of advice to every physician's wife in Texas. She urged each woman to support the American Medical Education Foundation, established in 1950 by the American Medical Association for aiding in and maintaining the expansion of the nation's 79 medical schools. Other projects mentioned were civil defense, nurse recruitment, dissemination of information about voluntary health insurance, school health programs, health days, health institutes, and mental health. She asked members to participate in all voluntary health organizations in the community the special emphasis of which is research, prevention, care, and rehabilitation, and to make a survey of health facilities and needs.

The Auxiliary is unique in that it is married to the medical profession, Mrs. Turner said, and has access to authentic sources of information. Doctors' wives can interpret the policies of the medical profession and make things happen in their communities which will make them better places in which to live if they will work under the direction of their county medical societies in all local organizations to promote health education and improve health facilities.

There being no other business, Mrs. John H. Wootters, Houston, moved that the meeting adjourn.

The day was ended with a dinner and an entertaining program at the Driskill Hotel Thursday evening. Mrs. S. M. Hill, Dallas, gave the "Highlights of My Trip to Europe," illustrated by slides shown by Mrs. S. F. Harrington, Dallas. Mr. Jack Darrouzet, Austin, captivated his audience with a "Bit of Magic." Dr. George Turner, El Paso, President of the Texas Medical Association, brought greetings and information concerning the care and cost of nonservice-connected illness of veterans, and the Bricker resolution. He urged the Auxiliary to take active interest in these fields. He gave briefly the Association's projects in its intraprofessional relations as well as its relations with the public.

MRS. PAUL BRINDLEY, Galveston,
Recording Secretary.

AUXILIARY STAFF MEMBER NAMED

Mrs. Arleen Draker has been named to handle business of the Woman's Auxiliary to the Texas Medical Association coming to the central office of the Association. A graduate in journalism of the University of Texas, Mrs. Draker has been an editorial assistant on the staff of the TEXAS STATE JOURNAL OF MEDICINE for the past year. All communications relating to Auxiliary activities should be addressed to Mrs. Draker.

CONFERENCE OF STATE PRESIDENTS

The tenth annual Conference of State Presidents and Presidents-Elect and National Committee Chairmen sponsored by the Woman's Auxiliary to the American Medical Association will be held at the Hotel LaSalle in Chicago, November 18-20. Dr. Franklin D. Murphy, chancellor of the University of Kansas, and Dr. Edward J. McCormick, President of the AMA, will be among the speakers. Mrs. George Turner, El Paso, President-Elect of the National Auxiliary, will preside over the conference.

DEATHS

H. A. BRIGGS

Dr. Harry Arthur Briggs, Albany, Texas, died June 15, 1953, in that city of a heart ailment.

Born April 2, 1888, in Wichita, Kan., the son of Joseph and Nellie Briggs, Dr. Briggs received his preliminary education in Palestine and at the University of Texas, Austin. He was graduated from the University of Texas Medical Branch, Galveston, in 1913 and interned at the St. Louis Maternity Hospital, St. Louis. He did postgraduate study in obstetrics at Johns Hopkins University School of Medicine. After serving as a captain in the United States Army Medical Corps during World War I, Dr. Briggs practiced in Wisconsin and Indiana before moving to Texas in 1942. At one time he was division surgeon for the Southern Pacific Railway at Corpus Christi.

A member of the American Medical Association and the Texas Medical Association, Dr. Briggs had belonged to the Nueces, Ector-Midland-Martin-Howard-Andrews-Glasscock, Rusk, and Taylor-Jones Counties Medical Societies. He was once secretary of his district medical society. He was a Mason, a Shriner, and a member of the Episcopal Church.

Dr. Briggs married Miss Marguerite Smith in Corpus Christi. She survives as do four children, Bryce Briggs, who is serving in the United States Navy at the Naval Air Station in Corpus Christi; Bill Briggs, Albany; Lt. Richard Feiden, stationed in the Philippines with the United States Air Force; and Mrs. Dawn Thompson, Mobile, Ala. Also, a brother, Frank Briggs, Victoria, and a sister, Mrs. John Meredith, Aransas Pass, survive.

M. E. TABER

Dr. Martin E. Taber, Dallas, Texas, died in a Dallas hospital August 24, 1953, following a short illness.

Born in Cairo, Ill., in 1875, Dr. Taber attended the Marion-Sims College of Medicine, St. Louis, from which he received his medical degree in 1898. Before beginning his practice in Dallas in 1904, he studied in Dallas and New York and also in Vienna, Berlin, and other European cities. Dr. Taber had been retired for approximately twelve years. His specialty was eye, ear, nose, and throat.

DR. MARTIN E. TABER

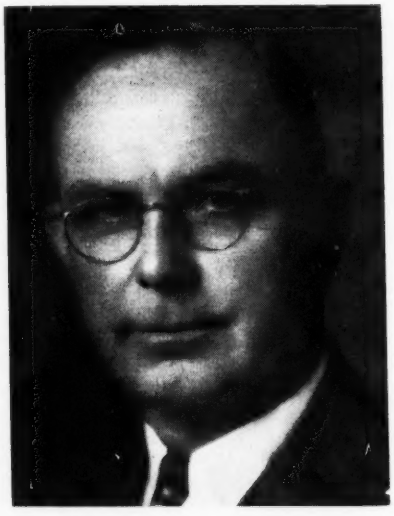
Dr. Taber, who served as Vice-President of the Texas Medical Association 1919-1920, was elected to honorary membership in the Association in 1947. He was chairman of the Section on Eye, Ear, Nose, and Throat for the Association's 1916 annual session. He was a member of the American Medical Association and the Dallas County Medical Society and was certified by the American Board of Otolaryngology. He was a thirty-second degree Mason, a Shriner, and a member of the Methodist Church.

Survivors include two daughters, Mrs. Warren J. Collins and Mrs. John F. Moore, both of Dallas; a sister, Mrs. W. W. White, Chester, Va.; a brother, R. G. Taber, Atlanta, Ga.; and four grandchildren, all of Dallas.

R. L. RAMSDELL

Dr. Robert L. Ramsdell, Norman, Okla., formerly of Dallas, Texas, died July 5, 1953, in a hospital at Ardmore, Okla., of cerebral hemorrhage.

Born in Salado, Texas, March 18, 1882, the son of Charles Henry and Augusta (Halley) Ramsdell, Dr. Ramsdell received his education at the University of Texas in Austin and the University of Texas Medical Branch, Galveston, from which he was graduated with a doctor of medicine degree in 1913. He then practiced in Houston for two years, San Augustine ten years, Dallas seventeen years, Austin nine years, and Norman, Okla., from 1952 until his death. While in Austin Dr. Ramsdell served on the staff of the State Hospital, and in Norman he was psychiatrist at Central State Hospital. While a medical student, Dr. Ramsdell spent one summer working with William Gorgas at the Panama Canal. Later he became especially interested in deficiency diseases, making a special study of pellagra.



DR. ROBERT L. RAMSDELL

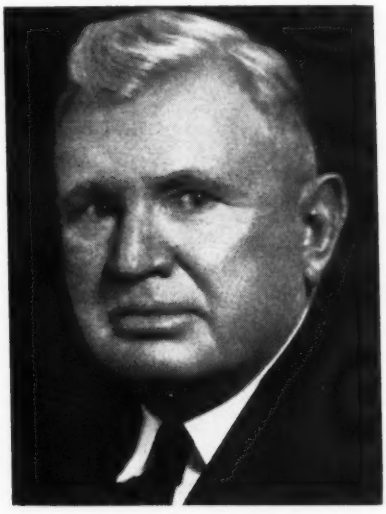
A current member of the American Medical Association and the Texas Medical Association through the Dallas County Medical Society, of which he was president in 1936, Dr. Ramsdell also was a past worshipful master of the Masonic Lodge in Dallas. He was a charter member of the Dallas Southern Clinical Society and a member of the Christian Church.

Dr. Ramsdell's wife, Mrs. Addie Hill Ramsdell, whom he married September 15, 1914, in Austin, survives him, as do a daughter, Mrs. J. W. Bridgewater, Akron, Ohio; a sister, Mrs. Ray Adamson, Glendale, Calif.; and two brothers, Dr. M. A. Ramsdell, San Antonio, and Fred L. Ramsdell, Philadelphia.

H. R. DUDGEON, SR.

Dr. Howard Rush Dudgeon, Sr., Waco, Texas, died in that city of coronary occlusion July 31, 1953.

A past president of the Texas Medical Association, Dr. Dudgeon was born November 21, 1873, in Chamois, Mo., the son of William T. and Mary (Wagner) Dudgeon. As a child, he moved to Texas, where he attended primary schools in San Marcos, was graduated from Coronal Institute at San Marcos, and attended Southwestern University at Georgetown before receiving his medical degree from the University of Texas School of Medicine in 1899. After serving as assistant surgeon of the Mexican Central Railway at Aguas Calientes, Mexico, Dr. Dudgeon interned at the John Sealy Hospital, Galveston, and was a demonstrator of surgery for two years before entering private practice in Ardmore, Indian Territory (now Oklahoma). He returned to Texas in 1903, after practicing a short time in Denver, Indian Territory, making his home in Lockhart. For the next ten years Dr. Dudgeon was on the staff of the University of Texas School of Medicine and at the time of his retirement from teaching was assistant professor of genitourinary diseases and dermatology and adjunct professor of surgery. He then opened private practice in Waco, where he was a surgeon until his death.



DR. H. R. DUDGEON

Dr. Dudgeon had been a member of the Texas Medical Association since 1904. He served the Association as chairman of the Section on Surgery twice, councilor for District 12, chairman of the Section on Obstetrics and Gynecology, President in 1936, and a delegate to the American Medical Association for about twelve years. He belonged to the American Medical Association and the Southern Medical Association and served as president of the Galveston County, the McLennan County, and the Central Texas District Medical Societies. He was a fellow of the American College of Surgeons and a charter member of the Texas Surgical Society.

During World War I, Dr. Dudgeon was a major in the United States Army Medical Corps and commanded a base hospital. He was a thirty-second degree Scottish Rite Mason, a steward in the Methodist Church, a former member and chairman of the school board at Waco, and active in political affairs.

Dr. Dudgeon is survived by his wife, the former Miss Sue Montgomery, whom he married at Maxwell June 11, 1903; a son, Dr. Howard R. Dudgeon, Jr., Waco; two grandchildren, Elizabeth Ann Dudgeon and Howard Dudgeon, III, Waco; a sister, Mrs. Emma Dudgeon Collins, Dallas; and a brother, Will Dudgeon, Tucson, Ariz.

V. M. LONGMIRE

Dr. Victor Melton Longmire, Temple, Texas, died at his home July 27, 1953, of heart trouble.

Born November 2, 1889, in Glenfawn, Dr. Longmire was the son of Dr. R. B. Longmire and Clara (Barber) Longmire. After being graduated from Jacksonville College in Jacksonville, Dr. Longmire attended the University of Louisville School of Medicine, Louisville, Ky., from which he received his medical degree in 1914. Since 1915 he had been associated with the Scott and White Hospital and Clinic, Temple. During World War I he was a first lieutenant in the United States Army Medical Corps, and he served as an examining physician in the United States Selective Service System from 1940 to 1947. His specialty was diagnosis and internal medicine.

Dr. Longmire was a member of the American Medical Association and the Texas Medical Association through the

DR. VICTOR M. LONGMIRE

Bell County Medical Society for many years. He was preceptor of the American Board of Internal Medicine, a life member of the American College of Physicians and the Masonic Lodge, and a member of the Shrine, American Legion, and Phi Chi medical fraternity. He was secretary of the Section on Medicine and Diseases of Children of the 1928 annual session of the Texas Medical Association and secretary of the Bell County Medical Society in 1938.

Married to Miss Ruth Rather December 3, 1923, in Temple, Dr. Longmire is survived by his wife; a daughter, Mrs. Willard B. Wagner, Jr., Houston; his stepmother, Mrs. R. B. Longmire, Hale Center; a brother, Barber Longmire, Jacksonville; two sisters, Mrs. E. H. Perry, Plainview, and Mrs. Rebecca Miles, Arlington; three grandchildren, W. B. Wagner, III, Alicia Longmire Wagner, and Julia Melton Wagner, all of Houston; and a nephew, F. L. Collins, St. Louis.

R. E. CROMEANS

Dr. Randall Elias Cromeans, Mexia, Texas, died of a heart attack July 7, 1953, in a local hospital.

Born in Fulton, Itwamba County, Miss., May 5, 1885, Dr. Cromeans was graduated from Oakland Institute, where he taught for ten years, and the University of Tennessee College of Medicine, Memphis. He began his medical practice in Tippah County, Miss., and then moved in 1915 to Strawn and in 1923 to Mexia, where he remained until his death. For fifteen years he was city health officer in Mexia. He served as a first lieutenant in the United States Army Medical Corps in California and France during World War I.

A member of the American Medical Association and the Texas Medical Association through the Limestone County Medical Society, Dr. Cromeans was also a member of the American Academy of General Practice, the Lions Club, the

DR. R. E. CROMEANS

Masonic Lodge, and the Baptist Church. He was president of the Limestone County Medical Society in 1939 and 1940 and secretary in 1942 and 1943.

Dr. Cromeans married Miss Mayme Vestal in Mart December 31, 1942. She and the following survive: a son, John Sharp Cromeans, Louisville, Ky.; three daughters, Mrs. W. G. Mask, Baytown; Mrs. E. J. Oates, Austin; and Mrs. Sam Sprayberry, Tremont, Miss.; a brother, Dr. Claude Cromeans, Belmont, Miss.; and six sisters, Mrs. Luna Davis, Mrs. Seth Storment, and Mrs. Loris Long, all of Belmont, Miss., Mrs. Eddie Frost and Mrs. M. A. Shelton, Birmingham, Ala., and Mrs. Glenn Greenwood, Fort Worth.

G. C. FARISH

Dr. George Clayton Farish, Houston, Texas, died July 1, 1953, of a heart disease in a Houston hospital.

Born December 15, 1894, in Sidon, Miss., Dr. Farish was the son of Emma (Moseley) and George Matt Farish. After attending Sidon public schools and the Agricultural and Mechanical College of Mississippi, Dr. Farish received a

bachelor of science degree in 1922 from the University of Mississippi, University, Miss., and a medical degree from the University of Tennessee College of Medicine, Memphis, in 1925. He studied at Tulane University of Louisiana School of Medicine, New Orleans, and spent twenty-two months specializing at the Eye, Ear, Nose, and Throat Hospital, New Orleans. He was president of the eye, ear, nose, and throat section of Jefferson Davis Hospital, Houston, in 1941. At the time of his death, Dr. Farish was emeritus professor of clinical otolaryngology at Baylor University College of Medicine, Houston, and an eye, ear, nose, and throat specialist with the Houston Clinic.

A member of the American Medical Association and the Texas Medical Association through the Harris County Medical Society, Dr. Farish also had belonged to the Ouachita Parish Medical Society in Louisiana. He served for three years in the United States Army during World War I. Dr. Farish was a member of the Catholic Church.

DR. GEORGE C. FARISH

In New Orleans Dr. Farish married the former Mrs. Bonnie Worley Martin, who survives him. Other survivors are a son, Robert Van Farish, Houston; two daughters, Mrs. James R. Shelley and Mrs. Norma Jean Scherer, Sacramento, Calif.; and two grandchildren, Bonnie Jo Scherer and May Jo Shelley, Sacramento, Calif. Several sisters and brothers from Greenwood, Miss., also survive.

E. R. LOCHTE, SR.

Dr. Erwin Richard Lochte, Sr., San Antonio, Texas, died July 30, 1953, in that city.

Born in Fredericksburg, Texas, in 1894, Dr. Lochte was educated at Austin High School and the University of Texas, both in Austin. While attending the University of Texas Medical Branch, Galveston, from which he received a doctor of medicine degree in 1922, he was manager for a year of the medical school's section of the "Cactus," yearbook of the University of Texas. He interned at St. Mary's Infirmary in Galveston and served in the United States Navy Medical Corps during World War I. He had practiced in San Antonio for thirty years.

A member of the American Medical Association and the Texas Medical Association through the Bexar County Medical Society, Dr. Lochte also belonged to the Texas Academy of General Practice, the Southern Medical Association, the International Medical Association, the Masonic Lodge, Consistory, and the Shrine, the Oak Hills Country Club, Fiesta San Jacinto Association, Phi Beta Pi fraternity, Order of Hermann Sons, Turner Club of San Antonio, and the Lutheran Church. He served as grand lodge physician for Hermann Sons from 1936 to 1949.

Dr. Lochte's wife, Mrs. Ellen Lochte, survives. Other survivors are a daughter, Mrs. E. M. Gregorie, Jr., Tucson; a son, E. R. Lochte, Jr., San Antonio; a sister, Mrs. Olivia Striegler, Fredericksburg; and three grandsons, Tucson.

H. W. COCHRAN

Dr. Harry Walton Cochran, Dallas, Texas, died July 28, 1953, at his home following an illness of three years.

Born in Belton, February 18, 1904, Dr. Cochran was graduated from the University of Texas, Austin, and received his medical degree from Johns Hopkins University School of Medicine, Baltimore, in 1928. He interned in medicine for four months at Baltimore City Hospital, Baltimore, and in surgery for sixteen months at Peter Bent Brigham Hospital, Boston. He was a junior fellow in surgery at Presbyterian Hospital in New York for a year and senior fellow the following two years. Since 1934 Dr. Cochran had lived in Dallas, where he was on the teaching staff of Baylor University College of Medicine, until 1943 when he became clinical professor of surgery at Southwestern Medical School. He retired in 1950 as a result of ill health. He was chief of the Baylor Hospital surgical staff from 1944 to 1950.

Dr. Cochran was elected to honorary membership in the Texas Medical Association in 1952. He also belonged to the American Medical Association, the Dallas County Medical

DR. H. WALTON COCHRAN

Society, the Society of University Surgeons, the Texas Surgical Society, and the Northwood Club. He was a past director and president of the Dallas Southern Clinical Society, was secretary of the Section on Surgery of the 1946 annual session of the Texas Medical Association, and was certified by the American Board of Surgery.

Survivors include his wife, Mrs. H. Walton Cochran; a daughter, Agnes Cochran; a son, Henry Thomas Cochran, all of Dallas; and a brother, Tom W. Cochran, Buckholts.